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EYE CONDITIONS THAT CONCERN THE PRACTITIONER

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In dealing with my subject, I assume that it will be agreeable if I confine myself largely to suggestions for the practitioner of medicine that may be of use to him in advising patients who may have to consult eye specialists. Patients are sometimes the victims of the overenthusiasms of specialists, and not the least among specialists are ophthalmologists. The practitioner who can protect his patient against the imbalance of specialism, advising him wisely, is rendering a valuable service. The specialist must be enthusiastic in his branch of medicine, and the non-specializing practitioner must see to it that the specialist keeps his balance and keeps his place.

REFRACTION

Not many years ago, a well-known oculist said that his idea of heaven was a place where there were no errors of refraction. Lack of proper training and lack of proper interest in refraction led him to this attitude. Every estimation of a patient's refractive condition has speculative interest for the oculist, and such interest should never be allowed to lag because of superabundance of material. Judgment, often a fine sort of judgment, is called for in advising patients who manifest errors of refraction. Too many people are wearing glasses. Instead of wise and thoughtful advice on the part of the ophthalmologist, overenthusiasm, error of judgment and mechanical ease of prescribing, contribute to this state of affairs. On the part of the optician, supreme interest in receipts has an influence. The result is that any oculist who practices a scheme for removal of glasses from patients has a fertile field. All oculists should be in the habit of ordering the discontinuance of unnecessary glasses.

Lenses should be prescribed either to furnish better vision or to relieve symptoms, or both. Working out problems in refraction, coming to decisions as to whether or not patients should wear glasses, figuring out what lenses good judgment would allow in case of necessity for the patient's wellbeing, wise counsel for the patients and their physicians—this is interesting and important work. There are patients who have the idea that they will "ruin" their eyes if they don't wear glasses. Seldom is a patient's failure to wear glasses responsible for a serious eye condition, so I think it is fair to patients that they should feel free in a general way to follow their inclinations or their own judgment in the amount of use that they make of glasses, depending on how much gain is furnished by them.

In hyperopia (farsightedness) all rays of light entering the eye come to a focus behind the retina unless they are brought to a focus by use of the accommodation or by use of lenses or both. Hyperopia is usual and normal in childhood. As the child grows the eye enlarges and hyperopia decreases. If there is not a good reserve of hyperopia it may be entirely lost during growth and myopia may develop. In my opinion it is absurd for the oculist to order glasses for a child simply because he has considerable hyperopia and by the oculist's theory may be having eye strain. Most children can furnish the necessary accommodation without undue strain. To be sure, nearly all children are keen to wear glasses and they will give expression to any symptoms that they think necessary in order to get them, and the doctor who refuses to prescribe them must run the risk of becoming very unpopular with his little would-be patients. In dealing with children the doctor must estimate the proportion of genuineness in the expressed symptoms and the proportion of commendable fabrication or imagination. When, however, there is genuine strain with symptoms referable to it glasses may be prescribed for either temporary or long-continued wear with most satisfying results.

Astigmatism is a structural condition of the eye in which light rays do not come to a focus. In small amount it may cause symptoms such as fatigue or headache, and in large amount it may

cause impairment of sight as well. I certainly would not prescribe correcting lenses unless there is likelihood that the astigmatism is responsible for trouble. There are very few eyes that have no astigmatism.

Myopia (nearsightedness) calls for glasses to give good distant sight and should not be regarded as serious unless it exists in large amount. In this condition the rays of light from distant objects focus in front of the retina so the myopic eye may be thought of as an oversized eye. During the growing period the eye gets gradually slightly larger, so myopia increases with body growth. Inasmuch as the years of physical growth are school and college years, there is a natural tendency to associate increase in myopia with reading and study, but there is no convincing evidence that close use of eyes is responsible for increase in myopia. So, unless the nearsightedness is large in amount, discontinuance or restriction in reading should not be ordered as a rule. In other words, I should not advise interruption or limitation of one's mental development on account of a small or moderate amount of myopia. Myopia predisposes to detachment of the retina, but the likelihood of detachment is inconsiderable so I should not advise abridgement of one's physical development or physical welfare on account of it. There is a popular belief that nearsightedness disappears in old age, but this is not so. Elderly nearsighted people are prone to take pride in their ability to read without glasses and are willing to go without good distant vision rather than to wear glasses. Any myopic or highly astigmatic person can sharpen his distant vision by skillful narrowing of his palpebral fissure (squinting).

One sometimes hears of patients who have been "cured" of myopia, so it may be in place for me to say that we have no remedial scheme for making an eye smaller or for in any other way reducing myopia.

Strabismus frequently calls for advice to patients or more often to parents. When true squint exists, neglect is not in order. Too often parents are advised to leave alone babies with convergent squint with the assurance that "they will grow out of it." The belief that commonly infants are crosseyed has resulted from faulty observation, and the recovery that is so often apparent is from a pseudo-squint and not from a real one. In such cases the inner canthi are set widely apart, and the cornea are near the inner ends of the palpebral fissures so that very little sclera shows to the inner sides of the corneas. This gives the appearance of convergent squint when it does not exist, and it has nothing to do with the axes of the globes. When the bridge of the nose develops and the inner canthi are drawn relatively inward, more sclera shows to the nasal sides of the cornea and the apparently crooked eyes appear to straighten. To judge roughly as to whether eyes are straight or crooked the physician should learn to regard the relative positions of the corneal light reflexes in the two eyes. These reflexes are in approximately corresponding places if the eyes are straight, not so if there is a squint.

If a true strabismus exists it should have early attention with two chief objects. First, the vision should be developed and maintained in the two eyes. Second, binocular single vision, with fusion of images in the two eyes should be developed if possible. In order to accomplish the second objective early operation may be necessary and I am unorthodox enough to advocate that operation be performed as soon as it is evident that non-operative procedures will not suffice. If properly selected and properly performed only good can result. It is not necessary for me to point out the embarrassment, self-consciousness and sense of inferiority that crosseyed children have to endure, and I believe they should be saved all this if possible. Let me advise that expert care be put into operation early in all cases of squint.

CATARACT

Cataract is opacity of the crystalline lens. Opacity may occupy a lens in any proportion from an insignificant part in some cases up to the entire lens in others. The interference in vision depends not only on the amount of opacity but also on the position. In elderly people a few opaque spots in the periphery of the lens appear as a rule, and it is unfortunate that the term "cataract" is used for these. Much more often than not blindness fails to result, and no mention should be made of these to patients, although descriptive notes should be made of them. More significant than a few opacities is the development of myopia in senile cases. This results from swelling of the lens fibers and gives rise to the well-known "second sight" that enables old people to lay aside their glasses and leads to so

much boasting. Second sight means development of cataract, but not necessarily development to maturity and blindness before death interrupts the process. In cases of oncoming blindness from cataract the question comes to the doctor over and over again, "Is there any cure for cataract except operation?" or "Is there any way of stopping them?" Many suggestions have been made for local and general treatment to arrest or eradicate cataract, and there might be a reasonable difference of opinions as to their value. However, it will be fair for me to say that I am not convinced that there is any value in any of them. But it can be said with assurance that operative treatment of cataract is most satisfactory. Properly performed cataract extraction is one of the most beneficient things that surgery can offer. In most cases normal sight or nearly normal sight can be made to result, with almost no risk of accident. It can be said also that a patient with cataracts does not have to wait until he is blind before extraction can be performed with safety. I might add that senile cataract extraction should not be regarded as an easy procedure. It calls for skill and precision, but if these are exercised sight will almost surely result.

In case of congenital cataract fine judgment is sometimes required from the surgeon for a proper decision as to whether operation is best or not, for congenital cataracts are ofttimes incomplete. The doctor should bear in mind that loss of the power of accommodation follows removal of the cataractous lens, and in a young person such loss is important. Certainly not all congenital cataracts should be operated upon. But if operation is deemed necessary, when should it take place in congenital cases? I should say the earlier the better so that central vision will be allowed to develop before nystagmus starts.

GLAUCOMA

One can almost say that the time has gone by when acute glaucoma is diagnosed "neuritis and acute gastritis" or as "cold in the eye." This condition is a serious one and calls for prompt action. If it does not yield to strong eserine medication operation should be performed. Iridectomy will almost surely reduce the tension in the ordinary acute type, and if done soon enough it will restore useful vision. It must be added, however, that there is appreciable risk in operation for acute glaucoma as there is a possibility of intraocular hemorrhage if the vessels are sclerosed.

In chronic glaucoma there is a genuine difference of opinion as to the advisability of operation. The difference is so wide that some ophthalmologists consider every case of chronic glaucoma a surgical case while others are totally opposed to operation. Manifestly both extreme groups are wrong. I think the case can be stated very simply. In many patients ocular hypertension can be controlled indefinitely by the use of pilocarpine and eserine, and the tension can be estimated with reasonable reliability with the tonometer. As long as the tension can be kept within normal limits with medication, and as long as the patient holds his own in visual function, such medication should be continued, for operation could not do more. (Another factor should be mentioned: occasionally either eserine or pilocarpine causes such a severe reaction that one is forced to operate on account of it. If medications fail to control tension, operation should be resorted to.

What prognosis can be given if the surgeon is forced to operate? In a high percentage of cases normal or nearly normal tension can be attained and kept and the risk from operation is not great. However, a glaucoma patient cannot be dismissed after operation as one can never be sure that tension will remain controlled, and function must be safeguarded. Chronic glaucoma is an insidious process and it must be watched whether operation has been performed or not.

OCULAR MANIFESTATIONS IN PREGNANCY

That pregnancy is responsible for contraction of the visual fields from enlargement of the hypophysis has been demonstrated satisfactorily. In 1922 Finley of Havana reported that "during pregnancy there often occurs, as a result of the normal hypertrophy of the pituitary gland in connection with this state, a compression of the chiasm which manifests itself by changes in the visual fields in the nature of a bitemporal contraction, which varies in degree according to the amount of compression suffered, this depending on the corresponding amount of hypertrophy and on the anatomical peculiarities of the case which may favor or hinder this compression." There is no impairment of the central vision due to the hypophyseal enlargement and the fields are not contracted enough to interfere with the pa-

tient's getting about safely. The phenomenon manifests itself during the last few weeks of pregnancy and disappears after parturition. It occurs in primiparas and multiparas. While this condition is interesting, it probably is not of great importance in its immediate manifestations and it is believed that there are no unfortunate late results.

Rarely optic nerve atrophy unassociated with retinitis develops late in pregnancy. This is thought to be the result of retrobulbar neuritis caused by the toxemia of pregnancy. It may result in partial or complete pallor of the nerve heads with partial or complete blindness. With the knowledge that the hypophysis is enlarged late in pregnancy it may be well for us to have in mind the possibility that in rare cases the enlargement might be great enough to cause pressure atrophy of the chiasm and optic nerves.

Choroiditis, although not generally recognized as a manifestation of toxemia of pregnancy, I think should be given a place among the important eye conditions caused by the gravid state. I have seen several cases of well developed exudative inflammation of the choroid in pregnant women who were sound except for their pregnant condition. The choroid is a highly vascular structure, and pathological response to pregnancy toxemia should not be considered a strange thing. In choroiditis the prognosis as to vision depends upon the location of the lesion. The portion of the retina which corresponds to the choroidal lesion is robbed of its function. If the lesion is in the macula, which provides central vision, serious permanent impairment of sight will result; but if the choroidal inflammation is away from the macula, the resulting blind area will not be important. Fortunately, the choroiditis most often affects only one eye, so there is small chance of serious impairment of sight in both eyes. In cases of development of choroiditis in both eyes or in a patient's only good eye, induction of labor might be wise.

Spasm of Accommodation.—Rarely the pregnant woman may be alarmed by impairment of her distant vision due to over-action of the ciliary muscle, resulting in a spasm of accommodation, the equivalent of myopia. She sees well (perhaps better than normal) for reading but at a distance her sight is blurred, and it may be necessary for her to wear concave lenses in order to have good distance sight. Presumably toxic substances stimulate the muscles of accommodation to a condition of mild spasm. The condition may continue for a few weeks after delivery, but relaxation gradually returns without serious consequences.

Hemorrhagic retinitis is the best known eye complication of pregnancy and the most important. Its presence may carry heavy weight with the obstetrician in his decision to induce labor, both because of its danger to the patient's sight and because of its significance in regard to the general condition of the patient. Without going into technical detail, it may be said that there are two types of retinitis in pregnancy, one associated with acute toxemia and the other with chronic cardiorenal disease. The retinitis appears during the latter part of pregnancy, and may occur in either primiparas or multiparas.

Each case of pregnancy with hemorrhagic retinitis should be judged by itself, but it can be said that the development of retinitis is always of serious import, and usually indicates prompt termination of labor. It is not necessary for me to say here that the lives of both mother and baby are at stake. As to the mother's eyes it may be said that some damage always results from retinitis. Blindness either complete or partial in both eyes is the usual outcome if the condition is allowed to remain long. In some cases, however, the optic nerve and retina are not badly injured and useful vision may result. Fortunately, the development of retinitis in one pregnancy does not mean that it will necessarily appear in succeeding pregnancies.

Uremia Amaurosis.—While all obstetricians are familiar with hemorrhagic retinitis of pregnancy many do not know of the rarer condition of uremic amaurosis, quite a different sort of a process. In this condition the patient loses the sight in both eyes, but ophthalmoscopic examination reveals nothing abnormal. The blindness is due to the toxic effect of uremia on the brain cells and not to involvement of the optic nerves or eye structures. Inasmuch as the process is supranuclear the pupils react to light, both directly and consensually, although the patient is blind. If the patient recovers from her uremia the sight is surely restored, and in this regard the prognosis is quite different from that in albuminuric retinitis.

NORMAL DIET FOR DIABETICS*

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Miami Beach.

The question of the best diet for diabetics is an old subject, on which it would seem all variations have been played. There have been low carbohydrate diets, low protein diets, high fat diets, diets balanced according to chemical reactions between glucose and fatty acids and finally undernutrition and starvation diets. It would seem that no new combination could be found, but since the advent of insulin, we can add to the above list, high carbohydrate diets in diabetes.

Dr. W. D. Sansum was the first to advocate the higher carbohydrate diets with increased insulin dosage. His article was published in the Jr. A. M. A. Jan. 16th, 1926, and brought out the fact that this was a safe procedure which benefited the patient both mentally and physically. It was after talking to Dr. Sansum in July, 1925, that I first began using high carbohydrate diets in treating certain selected cases of diabetes and called these diets "Normal Diets" inasmuch as they approached more closely the diet consumed ordinarily by a normal individual.

With the proper regulation of the insulin dosage any amount of carbohydrate may be safely given to the most severe diabetic. Now the question arises, since this is so, why should not a diabetic have a normal diet?

No exact definition may be given for the term normal diet, but based on dietary studies it may be said that the minimum protein intake of ordinary individuals is certainly not less than one gram per kilogram of body weight and that ordinarily a normal person will eat from two to four times as much carbohydrate as fat. Chemical study of the nitrogen equilibrium of the body would indicate that one gram or even a little less than one gram of protein per kilogram is amply sufficient.

In order to plan a diet of the character suggested, one may start with one gram of protein per kilogram of body weight of the patient and add carbohydrate and fat in the proportion of two to one, in sufficient amount to make up the total calories required by the individual. The caloric requirement of a patient can only be determined by trial and the use of scales. The least number of calories which will maintain an individual at a normal or a desirable weight under existing conditions is the caloric requirement. This naturally varies among other things with the amount of activity to which the patient is subjected in his daily life.

As a concrete example of calculating such a diet, let us suppose the patient weighs sixty kilograms and requires about 1800 calories to maintain his weight. Of this total 240 calories will be supplied by 60 grams of protein, leaving 1560 to be supplied by carbohydrate and fat, in the proportion of 2 grams carbohydrate and 1 gram fat. Two grams carbohydrate equal 8 calories and 1 gram fat equals 9 calories, so that for each 17 calories there will be 2 grams of carbohydrate and 1 gram of fat. Dividing 1560 calories by 17 gives 92, the number of grams of fat and twice this amount, 184, the grams of carbohydrate. The patient would, therefore, have a diet of 184 grams of carbohydrate, 60 grams protein and 92 grams fat, with sufficient insulin to keep the urine sugar free and the blood sugar at a fairly normal level. The diet for the same individual under the most commonly used diet at the present time would be 60 grams carbohydrate, 60 grams protein and 150 grams of fat. As a matter of gastronomic desirability, I invite any one to try these two contrasting diets.

As a proof of the feasibility of such a diet for a diabetic at no greater cost and with subjective and objective benefit, I submit the history of the patient who has been followed longest and most carefully. Other patients have responded in like manner.

The patient was first treated in September, 1923. He was a college sophomore, 23 years of age. He had the diabetes for two years and on a carefully and properly regulated diet had maintained a fair weight and good health until a few months prior to coming to the clinic. He had recently been losing weight on the diet which would prevent glycosuria.

He entered the hospital September 12th and was discharged September 17th, at which time he was sugar free on a diet of 80 C-65 P-180 F and 30 units of insulin daily, with a body weight of 60 Kg.

He reentered the hospital in October with a severe case of tonsillitis and a moderate degree of acidosis. Following recovery from the illness, his tonsils were removed and he returned

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home December 15, 1923, on a diet of 65-60-160 and 34 units of insulin. He weighed 64 Kg.

He entered the research laboratory of Eli Lilly and Company as assistant in April, 1924, and since that time has been under the closest observation and had no less than 519 blood sugar tests from April, 1924, to June, 1926, and, I might add, with no resultant anemia even though a daily average of almost 2 cc. of blood was removed for two years.

In April, 1924, he weighed 62 Kg. His diet was 90 C-70 P-210 F, furnishing 2530 calories. His daily dose of insulin was 38 units.

May 19, 1924, his diet was changed to 87 C-63 P-210 F, equaling 2490 calories and his insulin dose increased to 42 units per day in order to bring his blood sugar to a lower level. This diet and dose remained constant until August 4, 1924, when the fat was reduced 10 grams because he had gained to 64 Kg.

The diet and dose remained the same from August 4 till October 14th, during which time the patient gained 3 Kg. On October 14th, 1924, his diet was changed by again lowering the fat 10 grams, so that his diet became 87 C-63 P-190 F, a total of 2310 calories. His weight was 67 Kg. He remained constantly on this diet for nine months, that is until July 14th, 1925. His weight remained practically the same. His insulin for eight months had been between 50 and 56 units per day, and during practically this whole period his blood sugar had been kept at a sub-normal level in an endeavor to see if "resting" the pancreas would ultimately increase the patient's glucose tolerance. It may be said that if there was any gain in tolerance as a result of eight months low blood sugar, it was negligible.

On July 13th, the diet was still 87-63-190, a total of 2310 calories; the total glucose 142 grams and the insulin 50 units daily. The next day the diet was suddenly changed to a high carbohydrate diet and the fat lowered in such amount as to give about the same number of calories as previously, namely, 2309. The protein remained the same. The diet given was 242 grams C-63 P-121 F; in other words, a two to one ratio instead of a one to two ratio of C to F, and contained 290 grams glucose. The insulin was increased 38 units in order to take care of the additional 148 grams of glucose, calculating that each added unit of insulin would take care of 4 grams added glucose. How nearly correct this was may be seen from the fact that the blood sugar remained the same for nine days, after which it began to diminish. As a result the insulin was gradually lowered until the blood sugar became stabilized at the level maintained on the original lower carbohydrate diet. In the course of two and one-half months, it was necessary to lower the daily dose of insulin 16 units in order to avoid hypoglycaemia and for a subsequent period of two and one-half months, the same diet and insulin dosage was maintained with the blood sugar at or below the normal level. During this period of five months the body weight did not materially change. The final insulin dose was 72 units per day. Apparently after being stabilized on a low carbohydrate diet and insulin for a period of fifteen months, there was a material gain in tolerance for glucose as a result of giving a high carbohydrate diet for two and one-half months. This gain, however, may have been more apparent than real. During this period the patient's mental outlook was vastly improved, and he felt much better physically.

Naturally, as with any innovation, such as this one started by Dr. Sansum, there will be certain questions and criticisms.

The question of cost due to the increased dose of insulin required may be answered by saying that the lessened cost of the high carbohydrate diet itself more than offsets the added cost of the additional insulin. In the case above cited, the added insulin per day cost 16.5 cents, while the high carbohydrate diet was 18.3 cents cheaper.

The danger of large doses of insulin may be next considered. It has been shown that with large amounts of carbohydrate in the diet, there is less fluctuation in the blood sugar per unit of insulin, so that actually the factor of safety is greater. It would seem to be true, however, that the symptoms of hypoglycaemia under these conditions are different from those usually described; namely, there is more mental confusion and less of the physical signs such as hunger, weakness and sweating.

The bulk of material to be injected when larger doses are given offers no objection as insulin containing 40 and 80 units per cc. is now available.

Personally, I would not advocate increased carbohydrate in the diet of any diabetic who can remain sugar-free and in even moderately good condition without the use of any insulin. I

would not recommend it in the majority of patients who are now doing well on one injection of insulin daily.

I would use the so-called "normal diet" in all intelligent patients, who now require two or more injections of insulin daily; and particularly do I deem this desirable in those patients in the active period of life, as their economic statis, to say nothing of their general wellbeing, is greatly benefited thereby.

I would use higher carbohydrate feeding with children for several reasons: they usually require more than one dose of insulin daily, they are more prone to acidosis under the higher proportion of fat in the usual diabetic diet, and they often surreptitiously obtain carbohydrate when this is too severely curtailed in their diet.

CONCLUSIONS

The advantages to be gained by a more nearly normal amount of carbohydrate in the diabetic diet are—

1. It approaches the normal metabolic requirements.

2. It produces a more healthful state of mind and body.

3. It is more convenient and no more expensive.

4. It satisfies the patient and does not make him an object of remark or pity.

Since diabetes is due to a deficiency of insulin, it would seem logical to supply the deficiency rather than curtail certain elements in the diet below a physiological level.

DISCUSSION.

Dr. R. H. Mooty, Winter Haven:

I certainly congratulate Dr. Walters on his very interesting paper. I shall not discuss it in detail, but I want to make some comment. Where we do not have access to a blood chemistry laboratory, it is very difficult to handle a diabetes case properly.

The common sense feature of the paper is what appeals to me. Why in the name of common sense does it take we men, and women, too, so long to stumble over a thing before we can see it? Why, when a man is suffering from diabetes, should the doctor be afraid to give him any more to eat than to just keep him going along? Why not put him back to be a normal man eating normal food?

Dr. Woodyatt recently in Cleveland at the Post-Graduate Assembly, had a man in the clinic who had been a metal polisher, earning $40.00 per week. He developed diabetes, and although he had been put back on his feet, he was not physically able to work at his former trade, so he was clerking at $26.00 per week. Dr. Woodyatt suggested probably it would be better to put him on more insulin with more carbohydrates with it and go back to his old job. If you try to get too much mileage in driving your automobile and cut the gas down, it overheats your motor; if you have it too rich, it chokes up. So it is a common sense proposition. Feed him what it takes to make a well man, *i. e.*, a well man and able to carry on by the use of insulin to keep the body chemistry going near enough normal to keep out of trouble.

Dr. M. J. Flipse, Miami:

I think Dr. Walters is to be very highly commended for bringing this important advance in the dietary management of diabetes to our attention. I agree that the diet more nearly approaching the normal diet is to be greatly preferred. There is, however, a very choice selection to be made of these particular cases. Dr. Walters mentions some of the criteria on which we base this judgment. The patient who can get along without a hypodermic injection of insulin and can be managed by diet is probably better off with the diet. The patient who can subsist on one injection of insulin in the morning and a slightly modified diet should get that one injection. There are also those cases of moderately advanced diabetes who require two or three injections of insulin daily. There is a fourth class I would feel the insulin dosage is so large compared to the tolerance of the individual, that the diet had better be even more rigid than our ordinary diabetic diet.

I should like to describe one of the most severe diabetics I have ever seen, and because it presents certain perculiar abnormalities in nutrition, I feel it will be of value to know about this case.

The patient, age 35, came to my attention two and a half years ago in practically a diabetic coma. Weight 79 pounds. She had had insulin with a very rigid diet, with resulting decrease in blood sugar and loss of weight. She was placed in the hospital, suffering from exophthalmic goitre, which was considerably toxic. The patient was put upon a very low carbohydrate diet following the method of Woodyatt, giving very high fats. She gained in weight to

110 pounds. The thyroid was removed under local and gas anesthesia. The patient made a very stormy recovery, but after passing the danger line, again was placed upon her dietary regime. Some time later, a tumor appeared in one of the breasts that had malignant characteristics. This was removed. This time she had a less stormy recovery.

The interesting feature is that through the two and a half years the patient has gained in weight from 70 pounds to 135, a very little short of her normal weight. The blood sugar has been permitted to remain slightly above the normal. We do not reduce the blood sugar until the percentage runs over .24 per cent. We have considered the patient in good shape if the blood sugar runs .15 per cent to .2 per cent. She is eating 35 grams protein, 25 grams carbohydrates and 85 grams fats, or 1,000 calories daily and has gained in weight.

Dr. Hendricks:

Dr. Walters' paper brings to mind a case of severe diabetes that I have been following for the past six years. The onset of the symptoms was sudden and followed an acute streptococcic tonsillitis. When first seen his glucose tolerance was but 30 grams per day. This was increased to 35 grams per day by rest, alkalinization, and the cleaning up of all focal infections. At this point it remained, and no amount of dieting or starvation would increase it. This tolerance would not permit of a very liberal diet, and the most careful calculation or balancing of the various dietary components could not increase his intake to a diet yielding over 900 calories per day without a resultant appearance of sugar in the urine. Suffice to say this was not even a maintainence diet, and so the patient continued to lose weight and strength, until finally the slow starvation had so weakened him that even the ciliary muscles had not the strength to accommodate for vision, and final termination of the case seemed not far off.

At this time insulin was introduced, and the patient was placed on a daily dosage of ten units. He was a man of intelligence and he soon learned to interpret his own symptoms and look out for hypoglycaemia. From time to time he increased his insulin dosage, and made his diet more liberal, with a resultant return of strength and gain of weight. I last heard from him about two months ago, when he reported to me that he was taking 75 units each day, that he had gained over thirty pounds in weight, and was able to do a full day's work without undue tiring. He eats a normal diet and does not stint himself on carbohydrates. Under the old starvation treatment, his outlook was hopeless. Now after six years he can look forward to a long span of useful and happy years, for he is a normal man save for his lessened insulin secretion.

Dr. Walters is to be thanked for this timely paper which brings to us so forcibly the idea of adequate feeding of diabetics. In reality, all that was accomplished by starving a diabetic was his early death. In hypothyroidism, we administer thyroid extract in such quantities as will completely counteract our patients' thyroid deficiency; in diabetes, let us follow the same plan, and administer insulin in sufficient dosage to allow our patients to live a normal dietary life.

THE SIGNIFICANCE OF RECURRENT VOMITING OR THE SO-CALLED "BILIOUS ATTACKS" IN CHILDREN.*

GEORGE L. COOK, M.D.,
Tampa.

The term "bilious attack" belongs rather to popular than scientific phraseology, nevertheless this significant statement on the part of many mothers deserves more than a hidden smile. Indeed it is a complaint which should always call for a very careful consideration, granting that it is often a passing digestive disturbance. On the other hand, it will often later lead to watchful waiting for the fatal end, following anxious days of active treatment without any apparent results. It is a vague term with a misleading sound of innocence, which may have a tendency to inspire an altogether unjustified feeling of security.

Vomiting of this type has symptoms so unusual in character, that it is often looked upon and treated as an independent disease. From observation and study of the peculiar symptoms of this type of vomiting, we have concluded that they are very little understood. Many theories have been advanced in explanation of the pathogenesis of recurrent vomiting. Many writers maintain that the affection is a manifes-

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tation of a "uric acid diathesis," others associate recurrent vomiting with acetoneuria and designate it as "acetonemic vomiting" on the ground that acetone is so constantly found in the urine. There is no evidence, however, as some of the same writers admit, that acetonemia produces the attack. Since acetone is seen in so many other affections, the condition has been pointed out as a true acidosis. The occasional occurrence of jaundice, has lead others to believe the liver is at fault. It seems a fit conclusion that recurrent vomiting is due to a toxemia, derived from absorption of toxic products, accumulated high up in the intestinal tract with an accompanying liver insufficiency.

As the liver seemingly plays an important role in recurrent vomiting, let us consider briefly its functions. Investigators find the clinical study of the activities of the liver a difficult problem, for it is an organ with manifold functions, and detailed knowledge of some of its normal physiological activities is extremely inadequate. The liver through the portal vein collects the blood from the abdominal viscera and naturally must contain substances not found in the general circulation. We have known since the time of Claud Berbard, the importance of the liver in relation to the storing of glycogen, its metabolism, and redistributing the carbohydrate again into the general circulation. The liver is known to be the seat of much of the intermediary nitrogen metabolism in the body. The liver is largely concerned with the formation and excretion of bile. Widal has suggested that the liver removes and destroys toxic products of cleavage of the proteins which are present in the portal blood. In the presence of liver insufficiency these products enter the general circulation. Many dyes, when injected intravenously, are excreted wholly or in part by the liver, methylene blue and mercurochrome are examples. We also have fairly conclusive experimental evidence that fibrinogen is produced in the liver, or under direct influence of the liver, therefore, having a direct influence upon the coagulation of the blood. The liver, it is thought desaturates fats.

At this point I wish to call your attention to a case, the symptomatology of which to me seems characteristic. H. S., age two years six months, was said to have had frequent "*bilious attacks*" with vomiting. The first attack dated back to the first month of her second year. The attacks had become gradually more frequent, the last ones occurring about every six or eight weeks; all attacks were similar in kind, differing only in degree. They were usually preceded by a prodromal period, lasting from twelve to twenty-four hours, marked by such manifestations as coated tongue, constipation, languidness, irritability, loss of appetite and occasionally some abdominal discomfort. The mother stated that she always experienced great difficulty in getting the child's bowels to move, the stools being light in color and of a strong odor. Vomiting then began; the first vomitus consisted of undigested food, sometimes from one or two previous meals, later mostly of mucous or finally bilious and some times brownish stained vomitus. Up until the present attack mother had not noted any blood stained vomitus. The vomiting was generally distressing with retching and occurred when anything whatever was swallowed. She stated that even a drop of water would "come back." There was very little history of the temperature being elevated, she had never taken temperature but thought that only once or twice had the temperature been very high. The thirst was marked, the abdomen "sunken in," the heart usually beat rapidly. Before the child recovered she always became very weak and lost a considerable amount of flesh. The attacks usually lasted from two to three days. The mother with few exceptions had acted both as doctor and nurse. The attacks were just a "bilious spell" and she had learned from experience just what to do, to cure them. For the present illness the doctor was called, because of six days of intermittent uncontrollable vomiting. The only thing that hastened the mother to call for help, was the vomiting of blood stained vomitus. With this exception, she did not appear much alarmed, but did express surprise that she had not been able to control the vomiting as quickly as she had during previous attacks.

On physical examination the child was found to be extremely prostrated, emaciated, eyes sunken and deeply circled, eye grounds negative, the sclera showed only a slightly yellowish tinge, mouth and tongue dry, breath strongly acetone. Tonsils were hypertrophied nodular and on light pressure exuded small amount of caseous material. The skin was pale, dry, but showed no evidence of jaundice. The thirst was distress-

ing. The heart rapid, pulse weak, abdomen scaphoid. The liver was palpable two and one-half inches below the costal margin. There was no abdominal tenderness, nor were there any masses felt, the spleen was not enlarged. The peristaltic movements of the intestines were entirely absent, and only heard slightly following prolonged massage. The reflexes were normal. There was no rigidity of neck. Mother had not been able to get a satisfactory movement of bowels although had given calomel and oil in repeated doses. This she thought strange, as before such treatment had brought distinct improvement. The urine showed a total acidity 210, four plus acetone, two plus diacetic acid with 4 plus indican. No albumin, no sugar, no bile, the miscroscopic examination was negative, except for a few uric acid crystals. The white blood count was 10,000 with 65 per cent polys. The spinal fluid was negative. During the following six days there was intermittent vomiting with 14 convulsions, more or less severe in nature. All purgation and irrigation failed to produce satisfactory evacuations of the bowels. The slight stools were very offensive and of a trashy appearance. The total acidity was reduced to 10, the acetone and diacetic acid disappeared from the urine, but a secondary nephritis became quite in evidence.

During the entire course of the disease there was no evidence of bile in the movements. The liver increased in size. Despite all efforts, there was very little indications of peristaltic movements of the intestines at any time. Thirty-six hours preceding death there was some abdominal distension. Death in these cases is always preceded by stoppage of peristalsis. At first, the intestine became paretic from toxemia, then paralysis sets in. Life is incompatable with intestinal paresis.

Owing to these significant factors, there must be some very close association between the peristaltic waves of the upper intestinal tract and liver activity. The higher in the intestinal canal the supposed toxemia occurs, the more rapid the fatal end, as the absorbability of the intestinal tract begins at the stomach and gradually decreases throughout, being least marked in the rectum. When there is an accumulation of toxic materials, in the small intestines, where the absorbability is greatest, there is naturally thrown into the liver, through the portal vein, large quantities of toxic materials which necessarily have their influence upon liver activity. Excretions begin to diminish. And as bile has a most stimulating effect upon intestinal peristalsis, therefore, simultaneously with decreased secretions, the peristaltic waves begin to diminish, with this liver insufficiency. Decided metabolic changes take place within the liver and body tissues, which prove to be profoundly toxic in nature, and if not corrected in the beginning will cause death.

A high degree of acidity or an acidosis is almost a constant companion of the recurrent vomiter. The cited case well illustrates this point, also reminds us of the fact that overcoming the acidosis does not always cure the patient. The high degree of acidity, the acetone, and the diacetic acid, disappeared from the urine.

The term acidosis designates a condition in which the acid substances are present in the blood in a quantity relatively so increased that the normal excess of alkali is diminished. Experimental acidosis can be produced in animals by the use of hydrochloric acid. In man acidosis depends upon the relative excess of acid produced in the body. These excess acids in most cases are represented by the acetone bodies and consist of beta-oxybutyric acid, aceto-acetic acid and acetone. The relative excess of acids in the blood may be due to an overproduction of acid bodies, a failure of the lungs and kidneys to excrete them in sufficient amounts or to a loss of bases, i. e., a depletion of the alkali reserve of the body. This phenomena is found more or less prevalent in the recurrent vomiter. Acetone bodies are present in small amounts normally in the blood and at once become markedly increased in starvation. They are still more increased in recurrent vomiting, because in these cases you are dealing both with starvation and a toxic process which also has a decided tendency to increase acetone bodies.

I think it is well to mention here that an acetonuria, i. e., heavy acetone in the urine may exist without any symptoms of acidosis. Acetonuria designates merely the presence of acetone bodies in the urine. Acidosis is characterized by a relative decrease in the alkalinity of the blood independent of the amount of acetone bodies which are being excreted.

It is certainly true that children are especially prone to the development of acidosis; is it be-

cause of the small reserve of alkali they possess, or is it due to a peculiar aptitude with which their organs produce the acetone bodies?

Assuming that the vomiting is precipitated by absorption of toxic products accumulated high up in the intestinal tract, with an accompanying liver insufficiency, we must consider briefly the etiological factors concerning themselves in this phenomena, for certainly it must be some pathological process with its accompanying toxemia hampering the normal physiological activities of these organs, otherwise we would not have the metabolic changes taking place as previously related.

Recurrent vomiting is only nature's warning to look for real trouble It is a warning that something has gone wrong in some part of the body. The trouble may not be in the stomach at all, it may be in the intestinal tract, and often is (chronic intestinal indigestion) the kidneys, gall bladder, pancreas, the appendix. Some focal infection is often found as diseased tonsils, abscessed teeth, or it may be the nervous system, or heart, or it may be that faulty habit of eating an unbalanced diet. It may be caused from emotional disturbances bringing about disordered bodily conditions which are masqueraded as indigestion.

In the care and treatment of the so-called bilious attacks or recurrent vomiting a few differential points should be borne in mind. There is a disease, namely appendicitis, that I mention first because it is dangerous if unrecognized. Recurrent vomiting patients seldom have any abdominal signs. We are all familiar with the symptoms of appendicitis. I would advise a most careful examination of the abdomen always. Acute indigestion—this form of vomiting is often termed by parents "bilious attacks." A careful history will usually show a gross error in diet. Meningitis often comes disguised as an innocent stomach attack.

In closing allow me to impress upon you the careful sifting of the recurrent vomiter, for it is nature's warning to look out for the real trouble. What folly it is to try to soothe a symptom and leave the cause untouched.

DISCUSSION.

Dr. F. Clifton Moor, Tallahassee:

I think this paper deserves some real serious consideration. In the first place, we ought to make a very definite definition or differentiation in this type of vomiting to distinguish it from all other cases. A good many authors discuss this recurrent vomiting under the terms of cyclic vomiting, the typical attacks recurring with more or less definite periodicity. This symptom complex undoubtedly occurs very early in life. Most cases seen begin during early infancy and tend to improve with age. Most of them in my own experience are from six months to eight years. During that time, they appear every four to eight weeks. I think the general opinion is that there is definite evidence of acidosis. It seems there is some metabolic fault which interferes with the proper handling of fats. With this definition of cyclic vomiting, the question of treatment resolves itself into two phases. First, during the attack, the one principal is the administration of fluid and glucose. Of course, oral feeding is useless, so it means fluid and glucose per rectum, under the skin intravenously, intraperitoneally, any way to get it in, because the outcome of the case depends on the success in preventing dehydration and keeping the glucose high.

Recently some of the authors have found that cases that did not respond to glucose and fluid alone were quickly relieved by glucose plus insulin, which carries out the idea that they are probably definitely ketogenic origin.

Second, during the interval between attacks the cases should be kept on a diet very low in fats. My own practice is to eliminate all butter, mayonnaise, etc., and to order a diet of skimmed milk, cereals, green vegetables and fruit.

Dr. L. L. Whiddon, Fort Pierce:

Under the cloak of discussion, I want to make three or four statements. I don't think there was a paper read here in the whole period of the assembly that is more valuable than this, and it was certainly brought out in a good way. I sat on pins, though, afraid he would make some statements I would have to cross him on, but he didn't. I want to make these three statements in regard to biliousness, acute indigestion, liver medicine, etc. Gentlemen, get those three things out of your vocabulary, for there are absolutely no such things on the face of the

earth. That is, the way we generally mean it. Of course, you are bilious when you have jaundice, because you have pathological bile in your system, but that is the only time. A mother will say her child is bilious, and the doctors are to blame for it. They are the ones who start this mess. They cause these people to say this and to say acute indigestion and liver medicine, "dynamiting" the liver, etc. If you will read some of Joslin's and Balch's works, and other men who stand out above most of us in America, you will find out that they have absolutely proved there is no such medicine as a medicine acting upon the liver. Joslin, from several hundred experiments, proved only one substance to act upon the liver, and that was oxgall and then he wasn't sure that it wasn't the introduction of the oxgall that caused the increase of bile collected instead of the increased action of the liver.

Dr. George L. Cook, Tampa, closing:

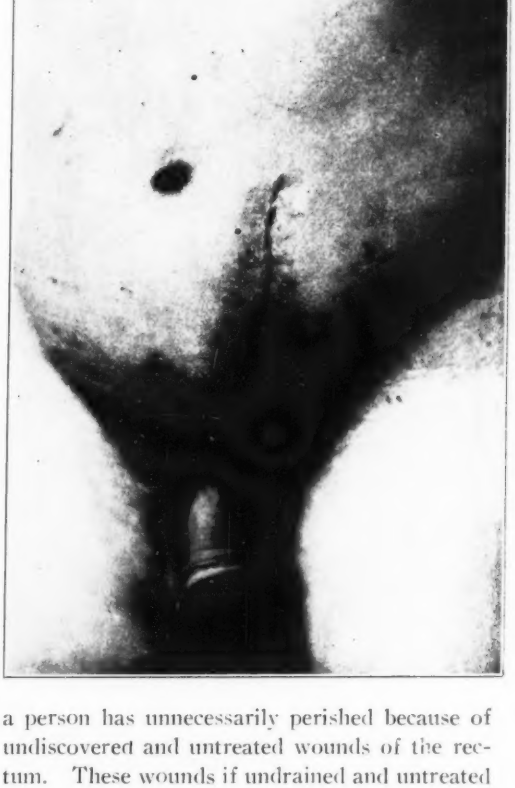
I was very much impressed with many points brought out in the discussions. In discussing a subject of this kind, you cannot cover the entire field. Recurrent vomiting, as I tried to impress, is a symptom, not a disease. I have been observing with particular interest these cases, and I have found they are handled usually as an independent condition. Recurrent vomiting is due to some toxic process going on in the body. The liver naturally collects all the blood from the intestinal tract. It has a function to perform that is certainly very difficult unless under the best conditions. If we have a case of recurrent vomiting, we pay little attention to the attack except to try to get the child to recover from the attack. You must locate the condition in the child that is producing the attack. It may be various things. I have seen a number of these cases cured by removing the tonsils, others by having infected teeth removed, but the majority are due to a chronic intestinal indigestion that exists in a large percentage of children with recurrent vomiting attacks. Also I have found a great many are due to drinking milk. I was interested in the paper regarding that subject. I am not ready just yet to state what percentage of children should not drink milk, but there are certainly from 3 to 5 per cent in which milk is absolutely contraindicated. I am for milk, I use it, lots of it, but there are certain cases that can not tolerate milk.

GUNSHOT WOUNDS OF THE RECTUM*

L. A. Peek, M.D.,
West Palm Beach.

Many times people are shot in the back, sometimes these wounds are low, often the projectile entering or passing through the rectum. These wounds may be overlooked and subsequent wounds, caused by the same projectile, such as wounds of the other pelvic and abdominal viscera, receive what attention is given the patient. There are also wounds, entrance points of which are from the front, the sides or from below that reach the rectum.

From my own experience I think that many wounds of the rectum are overlooked. When these wounds are not discovered and treated along proper lines the result is a fatality. Many



a person has unnecessarily perished because of undiscovered and untreated wounds of the rectum. These wounds if undrained and untreated frequently cause death. It seems that man is

*Read before the 54th Annual Meeting of the Florida Medical Association, West Palm Beach, April, 1927.

prone to think of forward things only and not recollect that there are backward parts too. So when a patient is brought to you with a gunshot wound you search diligently all through the parts of the pelvis and abdomen that can be reached by an anterior incision, you repair such wounds of various viscera that you discover, but the wound of the rectum that also happened at the same shot, because it is low and hard to find, you neither discover nor treat, and your patient dies. Only with the necropsy, if made, is the wound discovered.

Consider that any gunshot wound of entrance over any part of buttocks may wound the rectum. These wounds may sometimes be reached extraperitoneally from in front as in one of my own cases. They may also be reached from behind. After your exploratory laparotomy and such work as may be needed from the front is completed, you can close your peritoneal wound and go down from a left-sided laparotomy incision and keep outside the peritoneum and get down to where you can institute anterior drainage, and perhaps also repair the rectal wound. Or you can follow your wound of entrance, keeping thereby outside or rather below the peritoneum, enlarging the wound of entrance as needed.

The rectum is variable in position as any part of the alimentary tract. It may descend almost straight down from the sigmoid flexure, it may be a left-handed or a right-handed rectum. It may be full or it may be empty.

Case 1. Negro man, treated at Pine Ridge Hospital in 1915. Shot in back by policeman. Wound of entrance over mid-sacrum. Laparotomy done, several punctured intestines repaired. No wound of rectum found. Patient died on tenth day. Necropsy showed a punctured rectum with gangrenous stinking tissues postperitoneal of considerable extent. This had not been found at operation.

Case 2. White man, photographs shown. Shot in back by policeman, 1915. Entrance wound as shown in photograph. Laparotomy left external rectus muscle. Four punctures of intestines found and repaired. Peritoneum stitched with a drainage. Then the peritoneum was stripped down on left side and a wound of rectum was found, repaired and drained. The man made an uninterrupted recovery.

Case 3. Seen at necropsy by me in 1918 at Bellevue Hospital, New York City, where I was at that time stationed. The patient had been shot in back and attending surgeon had repaired the intestinal wounds through an ordinary laparotomy incision anteriorly made. But he had not found the rectal wound and the patient had died from that wound.

Moral—Always look out for gunshot wounds of rectum and be prepared to treat them adequately.

REPORT OF A CASE OF PREGNANCY WITH COMPLICATIONS*

HORACE STIMSON, M. D.,
Miami.

With some hesitation, for I do not wish to cast a pall over this happy occasion, I present the following case, not so much for your edification as for the sake of your advice.

April 18, 1927, Mrs. W. P. came to my office, referred by some friendly enemy, and cheerfully

*Read before the Dade County Medical Society, Miami, May, 1927.

unfolded the following story which I shall relate in chronological order. She was born 31 years ago in Iowa and her early youth was uneventful except for measles, pneumonia at 9 years, pneumonia at 18 and appendicitis at the same time, cured by the icebag. She suffered many attacks of tonsillitis, ending with tonsillectomy at 19. Menstruation was established at 14 and until marriage was marked by severe cramps before onset, requiring a "hypo" before each period. Marrying at the age of 21, 10 years ago, she soon afterwards fell downstairs bumping the end of her spine which has since been painful on sitting. Her husband has no complaints except that he was gassed during the War—and now works on the Causeway.

With pregnancy Mrs. P's troubles really began. She has had two pregnancies resulting in two healthy children aged 5 and 6 years. Both pregnancies were complicated by abscesses of the right breast which required lancing three times. During the first pregnancy she noticed, lateral to the left breast, a tender swelling which her Iowa physician diagnosed as an accessory breast; with lactation this tissue swelled to the size of the normal breast and was very painful. With each pregnancy she had vomiting and convulsions, severe occipital headaches, and was on a strict diet (presumably nephritic). With her second pregnancy she had heart failure in addition and was propped up in bed for two months, with swelling of all the limbs. Caesarean section was contemplated a number of times but her physician managed to carry her to full term parturition. Both labors were conducted by the Twilight Sleep method, aided by a general anesthetic, in hospital. The second labor was difficult, long, "dry," complicated by placenta previa and a foot presentation. Severe cervical and vaginal lacerations resulted, requiring suture. Both pregnancies were followed by severe hemorrhoids, the second by varicosities of the left leg; these have continued to increase in size and now cause dull pain and heaviness in the leg. Since the last pregnancy the injured coccyx has been so sore that she always has to sit sideways. She was unable to nurse either infant due to the breast abscesses. Since the last child she has gained from a weight of 145 to more than 200. She takes in washings, cares for house and family, and has no bad habits except indulgence in occasional hot toddies.

So much for her past story. Now her list of recent troubles begins. During the September, 1926, hurricane in Miami, where she has lived for the past two years, she was menstruating but the flow stopped, this being attributed to fright, drenching with cold water, etcetera. A month afterwards irregular frequent flow began and lasted several months, with gushes of blood and clots at times, a watery discharge at others. Early this year she suspected pregnancy and took whiskey and quinine. The flow continued and she went to a local physician who took her to a hospital February 18, 1927, 2½ months ago and, according to her story, scraped out the womb. She did not see him after the four days in hospital. Irregular frequent bleeding and watery discharge has continued. During the past month she has felt a quivering sensation low in the pelvis, the abdomen has enlarged, and she suspects pregnancy; there has been less bleeding in the past two weeks, but some watery discharge.

While being conducted to the examining room she stopped to tell me firmly that if pregnant she wished to "carry through" with careful supervision of pregnancy and labor; if not pregnant, she wanted a whiff of gas and the removal of the following items: the accessory breast, the loose coccyx, the left varicose veins, the womb trouble, and the tubes but not the ovaries.

With some trepidation I examined her and found an obese woman of early middle age, weighing 216 pounds; hair streaked with grey; a mass of breast tissue lying in the left pectoral region, measuring 5x5x3 inches, extending to the midaxillary line and quite distinct from the main left breast; a right chronic mastitis with three scars of old incision and drainage; heart and lungs apparently normal; blood pressure 118/75; a pregnant uterus with fetus lying in a left position and fetal movements strong; I should say she is six months pregnant. External pelvic measurements normal; no blood in vagina; varicosities of labia major; varicosities of left leg. The examination showed nothing else of note. She resisted catheterization but has promised to bring urine for examination.

Because a local physician in curettage left something in the uterus 2½ months ago, I find a six months' pregnancy on my hands, with the prospect of an exciting confinement—complicated by obesity, troublesome breasts, heart and kidney decompensation, eclampsia, varicose veins, and placenta previa.

Any suggestions from master obstetricians of Miami will be gratefully received.

PELVIC INFECTIONS.*

W. C. JONES, M.D.,
Miami.

In reviewing the literature of the past few years one is impressed with the great progress of medical science as compared with even a decade previously, too with the enthralling amount of research now under way, and the further need for study and close observation in order that the present great problems of medicine may be made simpler for future generations. This can be brought about, not alone through the aid of present day laboratory facilities but by the assistance of the general practitioner and specialist in taking complete histories, making thorough examinations, and studying the minor cases equally as the major. The skill in diagnosis is not tested so much by the major pathological condition, which usually has outstanding signs and symptoms, as by the delusive points in a persistent fever or uterine hemorrhage. The future of medicine, as brought out so well by McKenzie, depends upon the ability to correlate the beginning signs and symptoms of disease before there has been a marked destruction of the surrounding tissues, for gross pathology cannot be corrected.

In study of pelvic or gynecological disease there has arbitrarily been formed three major groups, and with few exceptions, practically all fall within these groups: namely, first, disturbed pelvic mechanics; second, tumors and growths, including ectopic pregnancy and early terminated pregnancies, and third, infections. The first two groups will not be discussed in this paper, as the last requires quite extensive discussion to be fully covered more so than time permits.

*Read before the Dade County Medical Society, May 6, 1927.

The literature is loaded with treatises on upper abdominal surgery, but comparitively very few have written concerning the pelvic diseases. Surgery of the pelvis has undoubtedly been abused and perhaps by no one instrument more than by the curette. Many a case of parametritis and fatal peritonitis has been brought on by the use of a curette and thus producing a wound infection. If any one tissue in the body acts as a guard and shield against infection it is the endometrium and why abuse nature and break down its defense in the face of eminent danger? The more delicate the tissue, the more frequent the infection and consequently the commoness of urethritis and the frequency of vulvitis and vaginitis in children.

A general consideration concerning the mechanism or causes of pelvic infection are: First, a direct canal extending from the vulva to the peritoneum; second, implantation of bacteria as gonococcus or through breaks in line of defense during labor or abortion and especially those brought on criminally; and third, the persisting latent infection in the deep cervical glands which becomes active as the result of opening up fresh channels for infection. These diseases are all the result of invading organisms and until the last few years the gonococcus was the most frequently active; however, in many localities the streptococcus is now running it a close second. The increased stress of living, the high cost of having babies, and often sexual immorality seem to account for the rapid increase of the latter. The illegal operation is usually performed by the illegal professionalist under adverse conditions or some person who introduces foreign bodies and creates an ideal field in the bloody mess for bacterial growth. In the obstetrical field through prenatal clinics, improved aseptic technique, and better facilities the puerperal sepsis has been markedly reduced. The frequency of infections due to abortions brought about by the irregular medical man or other means is used by advocates of birth control as a strong point in their favor. Other organisms which less frequently cause trouble are staphylococcus, pneumococcus and colon and tubercle bacilli. The infection by the colon bacillus is usually secondary to appendicitis or diverticulitis. The tubercle bacillus is not an infrequent cause but is rarely primary in the adnexa.

As a result of the above infections the condition may be local or general with a fortunate predominance of the former. The local condition may have varying constitutional symptoms with no blood infection, while the general condition often shows little or no local reaction but too often ends fatally with generalized peritonitis, or pyemia and its resulting multiple foci. The general effects will not be dealt with further in this paper.

The local reaction depends greatly upon the type of organism and the mode of introduction. The streptococcus which is usually introduced through a break in the birth canal is most likely to produce cellulitis or pelvic peritonitis. The gonococcus tends to produce urethritis, Bartholinitis, salpingitis, tubo-ovarian abscess, and often pelvic peritonitis. The colon bacillus may involve any of these structures. The tubercle bacillus most frequently infects the tubes. The infections may be mixed and the lesions multiple and complex. Although pelvic infection is very common, still no place in the body so readily localizes it as does the pelvis and undoubtedly its structure plays a great part in this function. It already has a floor and sides which are more or less fixed and only needs a roof to be completely shut off from the rest of the abdominal cavity. The tubes and ovaries have resistant walls and the pelvic ligaments, especially the broad, divide the cavity into sections.

Cellulitis usually involves the broad and sacro-uterine ligaments of one side but may be bilateral. Tubo-ovarian abscess is most often unilateral and associated with some local peritonitis. Salpingitis is commonly bilateral and the tubes may be converted into sacs or adhere to the ovaries to form a tubo-ovarian abscess and the pelvic peritoneum may be involved. In case of ascension of an infection from the genital tract after passing through the endometrium, the first line of defense, the highly convoluted mucous membrane of the tubes become rapidly congested and act as a block, while from contraction of the musculature the fimbriated extremities fold in and seal off or stick to some adjacent structure, usually the ovary. If, however, some of the infecting material seeps through into the pelvic cavity, it is low and soon nature covers over by agglutinating all available structures in layers to rapidly imprison the organisms, which causes the organisms to diminish their activity and reduces their virulence. In time they perish and meanwhile the patient is undergoing a process of self-immunization. The length of time necessary for this to take place varies with each case and is dependent upon the factors involved in the outcome of any infection, namely, virulence of the organism, resistance of the patient, and care and treatment used. Resolution gradually takes place but most often permanent damage has been done and acute exacerbations recur following the slightest indiscrimination or activity.

The onset of the infection is accompanied by pain, and frequently discharge and urinary symptoms. Pain is caused by the inflammatory congestion, discharge to the infection of the mucosa of the tract, and the urinary symptoms to urethral or perivesicular congestion and pressure. Nausea and vomiting may accompany it; temperature is elevated; and the lower abdomen is tender and rigid. For the first few days the above symptoms may increase in severity, but as localization occurs, the patient becomes more comfortable, and the temperature lowers with morning remissions. Convalescense is slow and may be accompanied by bad days. Leucocytosis is usually quite marked. The acute exacerbations most frequently occur at time of the menses and the menstrual cycle may be more frequent and profuse. If the condition does not tend to localize readily, constitutional symptoms are more severe with marked variation in pulse and temperature, chills, high leucocytes. There is most likely early suppuration which may need assistance.

The diagnosis of pelvic infection depends primarily upon a well-taken history and this will help find a post-abortive or puerperal starting point and also give evidence in the gonorrheal type of a discharge, urinary irritability or undue frequency in voiding urine. The symptoms accompanying the onset are very important and the examination is confirmatory. This examination should include a thorough investigation of the abdomen for any points of tenderness, masses, and any evidence of rigidity; the palpation of the abdomen should start over an unsuspected area for which the left upper quadrant is usually most suitable, and gradually work to the fields of tenderness and underlying pathology. The vaginal examination should begin with a stripping of the urethra, palpation

of Bartholin's glands and observation of vaginal walls and cervix. If any discharge is present, several smears should be made for microscopic examination—they being taken from urethra and cervix unless otherwise indicated. The bimanual examination should be made carefully and with extreme gentleness in order not to damage those cases where complete localization has not taken place. Occasionally anaesthesia may be necessary in order to successfully complete the diagnosis. A cellulitis produces a firm, fixed, immovable mass extending laterally from the uterus to the pelvic wall and quite inseparable from the walls. On the uterine side its upper limit does not go above the fundus, while further laterally it may produce a definite rounded mass above Poupart's ligament. The sacro-uterine ligament on the side involved is usually quite hard and thick. When the infection is unilateral the uterus is pushed to the opposite side, which may present quite a normal fornix. The uterus is quite firmly fixed but its size, shape and consistency are quite normal. This type of infection usually terminates without suppuration and may have no disabling effects. An ovarian abscess presents a definite rounded, elastic mass between uterus and pelvic wall with a definite sulcus intervening. Salpingitis presents masses in the lateral fornices, one or both of which may be in the sacro-uterine pouch. These masses are usually firm, fixed, and irregular. The lateral movement of the uterus is limited, although it may move freely in the antero-posterior plane. The tuberculous salpingitis gives quite similar findings, but onset is more insiduous and other tuberculous lesions are found in the body. This diagnosis may be more definite if self-immunization does not take place in six to eight weeks. In pelvic peritonitis the whole pelvis is more or less filled by a general inflammatory mass—the cervix is closer to the vaginal orifice; the fornices are filled and especially the posterior one which may show a definite bulging, fluctuant mass; and the uterus is not easily defined, being a part of the general mass formation. Absence of blood-infection, frequent chills with profuse perspiration, high temperature with marked fluctuations even to sub-normal, and tenderness over the course of the ovarian veins strongly suggest pyelophlebitis.

Differential diagnosis is most important in the case of appendicitis. History is important; the onset is more acute; nausea and vomiting more frequent; the pain is usually higher at first in the epigastrium and then lower in the right side with tenderness and rigidity over the appendix. Eliminating the association of this pain with recent labor or abortion or coincident menstrual period and determining the maximum tenderness to be over the appendix, the diagnosis of acute appendicitis is usually assured. A pelvic examination in any case of doubt is certainly not amiss. In cases of ectopic pregnancy the history and pelvic findings are usually quite definite. The recto-vaginal septum is not indurated as in case of inflammatory condition. Twisting of an ovarian cyst is not likely to be confused and an infection of a cyst is very uncommon. Fibroids are usually quite definite but differentiation from an old tubo-ovarian abscess closely adherent to the uterine wall may be quite difficult without a good history. In case of an infected or degenerating fibroid, in addition to the pelvic infection, there is usually a definite tumor and hemorrhage of marked degree.

Early treatment and management of pelvic infections should be conservative and non-surgical. Symptoms should be relieved and supportive measures followed. The patient should be put to bed and told to stay there for an indefinite period. This point usually governs the future course of the disease and financial condition of the patient becomes a great factor as to whether or not she can have proper attention. Even if she can be properly rested and cared for, she should be told of the process of self-immunization through which she must go and of the likelihood of good and bad days during this process. The local application of heat tends to relieve some of the pain and together with small enemas assist in keeping down distention. Hot copious douches of a mildly antiseptic solution except during the immediate onset assist in alleviating the pain. Morphia in small doses may be given hypodermically once or twice. For severe vomiting which is unusual, lavage may be used. The diet at first should be limited to plain fluids and gradually increased. After definite mass formation the diet may include the lighter foods and mild laxatives may replace the enemas. Every acute pelvic infection should be permitted to go through this period of self-immunization and convalescence. If her

status in society demands that she be returned as soon as possible to her household duties, or if the process is a menace to her future health, radical surgical procedure should be undertaken. Without operation, an early return to duties, lack of proper care, exposure to cold, dampness or fatigue may soon result in an acute exaverbation. In any case that has gone through the self-immunizing period and then follows with one or more acute exacerbations, there is proper reason for radical surgery. However this surgery should preferably be done, if circumstances permit, after at least two weeks of normal temperature. At this period the least destructive surgery is possible and even if small pockets of purulent material, which is usually sterile, escape during the removal of the pelvic masses, it may be carefully sponged away and an operative field free from hemorrhage and foreign products may be closed primarily without drainage. During my past hospital service, I operated twenty-three cases of chronic salpingitis, twelve of which showed purulent material during the operation. These fields were carefully freed from hemorrhage and all cases closed without drainage. The post-operative course in these cases was not atypical to the clean cases and all healed primarily with no evidence of local masses at end of two weeks' post-operative.

In the abdominal operative work it is well to be conservative but at the same time radical enough to eliminate all possible chance of future ill-health from pelvic trouble. Up to the age of thirty-five or even forty, if possible, both ovaries should be preserved as well as a healthy uterus, in order that normal menstruation may continue. If both ovaries are destroyed, it is a good practice to remove the uterus as well and either cauterize or cone out the cervix to eliminate future discharge. There are various aids to treatment such as vaccines, foreign proteins, dyes, and other agents, the merits of which will not be discussed in this paper.

If in treatment of pelvic infections nature's plan of defense and overthrow of enemy organisms are recognized and the course governed by same, the present mortality rate of many surgeons and hospitals will be remarkably lowered, for after all is said and done, the sooner nature is heralded as the best doctor and is assisted in her plans and methods, the sooner will humanity benefit and the physician's reputation augment.

MULTILOCULAR CYST OF OVARY IN THE INGUINAL CANAL—CASE REPORT*

S. E. Chambers, M.D.,
Miami.

Mrs. H. H., a white woman, 22 years of age, presented herself with the complaint of a mass in the left inguinal region of two years' duration, but enlarged markedly in the past six months.

The family and past history is essentially negative with the exception of a tuberculous disease of the left hip ten years previously which was operated upon by Dr. Albee with partial ankylosis and an apparent cure of the joint condition.

On physical examination the patient is slightly underdeveloped, but fairly well nourished. Both apices of the lungs reveal evidence of fibrosis but no rales. The left hip is partially ankylosed. In the left inguinal region there is a mass of about 8 cm. in diameter, firm, not fluctuating, skin over the mass freely movable. On coughing there is no impulse transmitted to the finger. It was my impression that I was dealing with either a sliding inguinal hernia which had become incarcerated, or a lipoma, the latter being of not uncommon occurrence in this region. On palpation I could make out a mass in the left iliac region. Its relation to the uterus could not be made out.

On opening the inguinal canal under one per cent novocain anesthesia, I came across a mass with thick walls corresponding to the mass that could be felt on the outside. The incision extended from the anterior-superior spine of the ilium to the symphysis, but the mass could not be delivered through the incision. Therefore, we divided the oblique muscles of the abdomen transversely after suturing the muscles to the fascia. The mass was delivered through the incision and was connected by a narrow neck which extended through the canal with a mass inside the peritoneal cavity about four times the size of the mass outside. The large portion of the tumor was anchored by a pedicle, rather short, and traceable down to the left ovary. We were dealing with an ovarian tumor which had herniated through the inguinal canal. There was no evidence of strangulation or twisting of

*Read before the Dade County Medical Society, August 6, 1927.

its pedicle. The tumor was multilocular and filled with straw-colored fluid.

The pedicle was ligated and the tumor was removed en masse. The wound was closed in the usual manner after the repair of the inguinal opening. The patient had an uneventful recovery and was discharged cured 15 days after the operation.

This case is here reported because of its comparative rarity, only two cases being reported in literature, and the importance of bearing this condition in mind in tumors in the inguinal region. Also, danger of strangulation by the twisting of the pedicle is a possibility, which will require immediate surgical intervention.

PNEUMOTHORAX IN A NEWBORN INFANT—CASE REPORT*

M. Jay Flipse, M.D.,
Miami.

The occurrence of pneumothorax during infancy is very unusual. Text books on pediatrics are either silent in regard to this condition or only mention its rarity. Considerable theoretical discussion is found on the physiology of respiration in infants, all of which indicates that there is at best only a very small negative pressure in the pleural cavity of the newborn.

Abt states that the lung of the newborn does not collapse if the thorax is opened, and that the negative pressure in the lungs during inspiration is probably not more than 5 mm. below atmospheric pressure. A search through the available current literature fails to reveal any reports on spontaneous pneumothorax in infancy, although the condition is by no means uncommon in adults.

Cozzolino in a search of the literature up to 1906 found forty-one cases of pneumothorax in children. Forty per cent of these were tuberculous. I have not been able to obtain his original article as yet and do not know the youngest child in his series.

Cattelan describes a case in a child of three years who developed bilateral pneumothorax as a complication during measles and pertussis.

To these cases we wish to add our own report. There is no question as to the diagnosis in our case, although the etiology is obscure. An excellent series of X-ray films was obtained by Doctor Raap, indicating the progress of the infant under treatment.

Mrs. G. O., age 22, white female, was delivered of an eight-pound female infant about 5:30 a. m. September 22, 1927, by Doctor M. D. Wilson. Labor had been induced by means of a Brown bag and the child was delivered with the application of low forceps. The infant was normally formed, robust and of good color on delivery. The fetal heart rate had been in the neighborhood of 130, before and during labor, and was approximately of the same rate immediately after birth. As soon as the child was delivered it made a vigorous attempt to breathe, but little or no air entered the lungs, the lower costal margin being deeply retracted during the inspiratory effort. The cord was ligated and severed, and ordinary measures of inducing respiration were used, such as spanking the child and gently compressing the thorax with one hand. When respiration failed to begin with these measures, a small tracheal catheter was passed. The compression of the chest was then continued, and air could be heard passing through the catheter. The infant also received a small dose of Alpha Lobelin hypodermatically. Within a few minutes respiration had begun and the moderate degree of cyanosis which had developed cleared.

Respiratory movements at this time appeared of an unusual type. Expansion of the chest was unequal, being apparently limited to the left side. There was, however, a sufficient oxygenation of the lungs to warrant withdrawal of the catheter. At this time the infant cried with a rather feeble but otherwise normal voice. It was noted, however, that on slight exertion, either with crying or on movement of the limbs, the child developed cyanosis. During these periods of cyanosis oxygen was administered, which restored the normal color.

Three hours after the birth of the infant I saw the child in consultation with Doctors Wilson and Kennon. At this time the expansion of the lungs appeared to be fairly equal, but the left side of the chest was more prominent than the right. Percussion note was resonant throughout, slightly higher pitched over the left than on the right. The heart sounds were heard more clearly to the right of the sternum. Breath sounds were of normal broncho-vesicular type over the entire right chest and were practically absent on the left except along the spine posteriorly where distant tubular breathing could be heard. A tentative diagnosis of pulmonary ate-

*Read before the Dade County Medical Society, October 7, 1927.

lectasis was made and X-ray of the chest was ordered. The X-ray films showed the left side of the chest larger than the right, and the heart displaced almost completely to the right of the sternum. The left lung appeared fifty per cent collapsed and a well defined area of pneumothorax completely surrounded the left lung.

Since the child appeared to get on satisfactorily by the use of oxygen during its occasional spells of cyanosis, we deemed it advisable not to interfere with the pneumothorax until a later time. Additional X-ray films were made at twenty-four hour intervals. These showed a progressive collapse of the left lung. On the morning of the twenty-fourth, the left lung was totally collapsed, and in spite of the fact that the clinical manifestations were improved, it was considered advisable to remove some of the air from the left thorax to enable the heart to return to a more normal position. Accordingly 30 cc. of air was permitted to escape by puncturing in the mid-axillary region with a ¾-inch, twenty-five gauge hypodermic needle connected to a twenty cc. luer syringe by a short length of rubber tube.

The infant showed no reaction from the aspiration of air, and films made immediately after this procedure gave evidence of some expansion of the left lung. The heart was also several cm. nearer its normal position, although the cardiac shadow was still displaced considerably to the right. There still remained a considerable area of pneumothorax on the left side. The following morning X-ray films showed the left thorax smaller than had previously been noted. The diminution in size, however, was at a sacrifice of lung expansion. The left lung had collapsed to nearly its former state; the amount of air in the left thorax was obviously decreased. Eighteen cc. of air was withdrawn from the left chest without suction, the air being forced out while the child was crying.

The nurse reported that the infant had shown a single attack of severe cyanosis and choking during the preceding two hours. Shortly after the second aspiration of air, the child regurgitated a small amount of breast milk. Immediately a very pronounced stridor developed and the child presented symptoms of obstruction of the glottis. These symptoms were relieved by inverting the infant and administering oxygen inhalations. For at least ten minutes after the obstructive symptoms were manifest, there was a marked stridor associated with retraction of the thorax and slight cyanosis. It was our impression that these symptoms were due to obstruction of the glottis, either by spasm or by collapse of the epiglottis.

The following day X-ray films showed the left lung in apposition with the chest wall except for a very small portion along the lower border. The heart at this time was in a practically normal position and the general condition of the infant satisfactory.

Before discharging the infant from observation, consultation was obtained with Doctor Hotchkiss to determine the condition of the epiglottis. On attempting to examine the throat the child vomited and developed a moderate degree of stridor, which, however, subsided without cyanosis. Doctor Hotchkiss was of the opinion that the cause of the stridor was collapse of the epiglottis. He advised that no measures be taken until observation indicated the degree of epiglottic collapse. Since that time the infant has progressed very satisfactorily, has shown no further abnormality of either the lungs or the larynx and is behaving in all respects like a normal infant.

The mechanism in the production of the pneumothorax in this case is problematic. It is our impression, however, that at birth the infant had a collapsed epiglottis which obstructed the larynx. It is altogether possible that the inspiratory efforts made at that time were sufficient to rupture an alveolus. The asymmetrical expansion of the chest when respiration had been established after the introduction of the trachial catheter suggests that the pneumothorax was even then in the process of development.

Collapse of the epiglottis, though rare, is occasionally observed. In a number of instances it has been necessary to amputate the epiglottis to prevent asphyxiation. In this case we are probably dealing with an epiglottis with but slightly more flaccidity than normal and which will probably develop sufficient cartilage to become rigid as the child grows older.

DISCUSSION.

Dr. W. T. Hotchkiss:

This is certainly a very interesting case. I think that Dr. Wilson and Dr. Flipse are to be congratulated on the way they handled the case at the very onset. It is undoubtedly true that the prompt introduction of the tracheal catheter saved the child's life.

Something we all ought to keep in mind is the

cyanosis. These cases of achondroplasia of the epiglottis are fairly rare, but the literature does contain a good many cases. In the average case where there is practically no cartilage in the epiglottis, the epiglottis should be very promptly amputated. There is no relief otherwise. This child must have had some cartilage in the epiglottis—there was probably a thin line through the epiglottis where there was no cartilage which produced the valve-like action.

Amputation of the epiglottis does not leave any bad results whatever. The children go on just as well as though they had an epiglottis. There is no interference in swallowing, which shows the epiglottis is an organ, after all, of no particular importance, and the larynx covers the opening preventing the inspiration of foreign bodies.

There is another condition that sometimes does exist that may be confused with the loss of cartilage or collapse of the epiglottis. There were about forty cases in the literature up to 1926 of mucus retention in the epiglottis. This forms a cyst of the anterior wall of the epiglottis and produces a constant dyspnea and cyanosis until relieved by collapse of the cyst and evacuation of its contents. In most of these cases, it is not necessary to amputate the epiglottis.

Whenever we have a case of cyanosis and stridor, we probably have an epiglottis disturbance of some kind, either cyst or achondroplasia of the epiglottis.

ETHER ANAESTHESIA*

J. M. Beggs, M.D.,
Chattahoochee.

Ether has been used as the principal anesthetic here since 1920, and in the last four years it has been administered 1,143 times, to patients from 2½ to 80 years of age, to all types of mental cases, and some few with valvular heart diseases, and practically all types of major operations and accidents, with only the following fatal results, which might have been due to the ether: One death from aspiration pneumonia, 3 days later; one death on table while doing an operation for strangulated hernia; one death shortly after completing amputation right leg at middle third femur, from an accident of log truck running over man. (About 2½ hours elapsed from the time of the accident until he reached the hospital and he was in quite a state of shock, so do not consider this death due to ether.) One death following thyroidectomy, about 35 minutes after leaving operating table, which might have been caused from the anesthetic. Out of this series of patients we have found only one in which ether was contra-indicated, that was due to patient having marked pulmonary edema.

We use ether for the following reasons: easily obtained and transported, economical, does not require cumbersome apparatus in administration, free from accidental explosions in the ordinary operating room, and probably does not require as much skill in administering as some of the others.

Preparation.—Usual preparation the night before for operation, and half hour before going on table hypo of ⅛ to ¼ gr. morphine sulphate and atropine in proper doses. Just before going on table all false teeth removed, should patient have any, face and mouth anointed with vaseline, gauze pads over eyes and towel over head.

Administration.—We use the open drop method, with Esmarch mask covered with 4 to 8 layers of gauze, and add towel around this. In beginning, hold mask about six inches above patient's face and give gradually, also lowering mask gradually. In this way, can get the majority of patients etherized without their becoming excited, also without having the sensation of strangulation. It usually takes from 10 to 20 minutes to get a patient ready for operation, taking from 6 to 8 ounces of ether, as it is not all utilized due to evaporation. After the patient is relaxed and towel placed around mask, decrease the amount of ether and try to give just enough to keep thoroughly relaxed. Keep the patient's head inclined to one side as they breathe better, and there is not as much chance of tongue becoming troublesome by falling back and obstructing breathing. Lots of times when patient's breathing is shallow and irregular a few drops of aromatic spirits of ammonia will put him back to normal.

Shock is best treated by prevention. The surgeon can practically eliminate shock in abdominal operations by careful and gentle manipulation of the viscera, and the anesthetist by watching the patient closely for the cardinal symptoms, such as cyanosis, rapid, irregular pulse, cold clammy skin, and dilitation of the pupils. If the

*Read before the Leon-Gadsden-Liberty-Wakulla-Jefferson County Medical Society, Chattahoochee, October 13, 1927.

shock is due to loss of blood, before or during operation, morphine by hypo and normal saline by hypodermoclysis or intravenously, is the best treatment, with the application of heat by hot water bottles. Outside of cases mentioned which were fatal, we have had only two patients to go into shock on the table—one was treated as mentioned above; in the other one, the operation was completed so the patient was put to bed. We have had few cases which went into shock following operations, which consisted of one gallbladder operation, and one pelvic operation.

In gallbladder operations it is necessary to have the patient thoroughly anesthetized; in fact, pushed near to where the pupils are partially dilated, as it is necessary to prevent shock afterwards. In other abdominal operations it is not necessary to give to the point of dilating the pupils. In pelvic operations, especially where tension has to be applied to uterus, the breathing is shallow and sometimes irregular, but as long as the patient's color is good and pulse is full and regular, and no dilitation of pupils, it should not cause any alarm to the anesthetist.

We have noted that the following are the hardest to anesthetize, and keep on what might be called even narcosis, in order named: alcoholics, drug addicts, and that type which might be described as short in stature, compact physique, and short fat neck.

TULAREAMIA—CASE REPORTS

C. W. Harper, M.D.,
Chipley.

Case 1—R. E. H., white farmer, age 45, in latter part of February, 1927, noticed three small pustules on right first finger which he opened with a knife used in dressing wild rabbits the day before. Three days later he developed a severe headache, pain in back and limbs, next had a chill, fever and sweats which continued about ten days. Two days after appearance of fever, right first finger was badly swollen but only slightly painful, the pustules had ulcerated and a thin purulent fluid could be expressed. About this time a small lump appeared on mid-forearm, a larger one on mid-arm and a mass in axilla, all very tender and painful. These glands were later incised and revealed thick creamy pus. Several small pustules appeared on chest and back. He lost twenty pounds and was unable to work for six weeks. Blood sent to Hygienic Laboratory, Washington, agglutinated Bacterium tularense in dilution of 1:1280.

Case 2—Mrs. J. H., white housewife, age 46, five days after killing and tearing up wild rabbit in her garden—this done to keep other rabbits out (?)—developed headache, pain in back and limbs and some temperature and much nausea and vomiting, the latter aggravated by the thoughts of a rabbit—this before the probable connection between the rabbit and her illness was suggested. On the right thumb two scratches which had been present for some time became inflamed and ulcerated, causing but little pain. Next day, on right arm, a red streak with several lumps along its course and in axilla appeared, all tender and painful, two weeks later these glands were incised and revealed thick pus. Recovery has been slow, patient very weak and nervous with soreness and stiffness of muscles and joints of back and limbs, seven weeks after onset of symptoms. Blood sent to Hygienic Laboratory, Washington, agglutinated Bacterium tularense in dilution of 1:320.

U. S. Public Health Service,
Washington, D. C.,
March 22, 1927.

(COPY)

Dr. C. W. Harper,
Chipley, Florida.

Dear Doctor Harper:

Three serums forwarded with laboratory slips were received March 21st and were tested for agglutination of Bacterium tularense with the following results:

R. E. Hood, positive, dilution..... 1:1280
Julia Hicks, " " 1:320
Lucy Everette, negative in all dilutions from 1:10 to 1:5000.

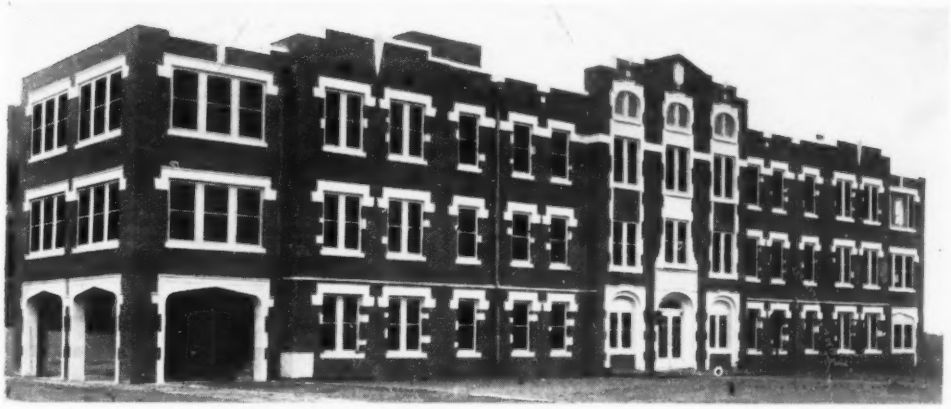
This is a remarkable group of cases and I congratulate you on the diagnosis. The serum of Lucy Everette was collected apparently on the eighth day which is on the border line of beginning agglutinin formation; if you will send this serum again it will doubtless be positive.

The diagnosis of Tularemia is like working a cross-word puzzle; the trick is to find the letters which spell R A B B I T.

Enclosed please find three questionnaires on which I would appreciate receiving the high points of the history.

We are ready at all times to test serums.

Sincerely yours,
Edward Francis,
Surgeon.



NEW MUNROE MEMORIAL HOSPITAL, OCALA

The formal opening of the handsome new Munroe Memorial Hospital was celebrated January 6th, when hundreds of Marion County citizens paid their visit to the institution.

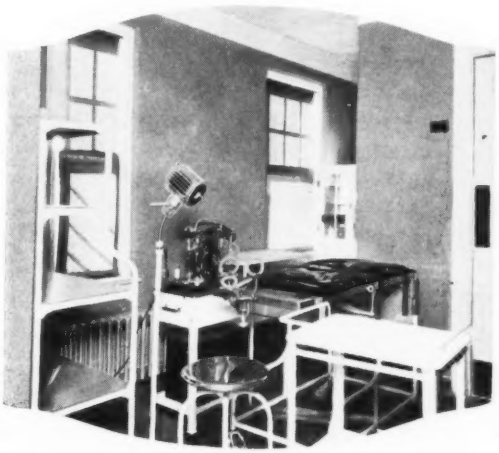
Nurses from the training school were stationed throughout the building acting as guides describing the equipment, and answering the numerous questions asked. The hospital seemed to have the unanimous approval of all, and numerous were the compliments passed to those responsible for the very fine equipment installed.

The hospital is a fire-proof, brick structure situated on the South Dixie highway in the center of a five-acre tract of ground with beautiful terraced lawns running to 12th street. No more ideal location could be found surrounding Ocala for a hospital. The hospital has a capacity for sixty-six patients and in an emergency could take care of approximately ninety patients. The top floor contains the major and minor surgery, sterilizing room, delivery room, doctors' scrub and dressing rooms, X-ray and orthopedic rooms. The rest of the floor is devoted to maternity cases, baby cribs and children's ward.

Public Ward.

The second floor is arranged for the care of surgical cases on one end and medical cases on the other, with the male and female wards on either side of central corridor. The main floor contains the administrative offices, doctors' staff room, emergency room, reception room, nurses' dining rooms with large kitchen, which is equipped with electrical apparatus. All of the equipment in the hospital is of the very latest design and no money has been spared in procuring equipment to render the very highest service to care for the sick and the invalid.

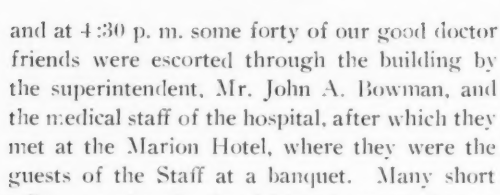
Invitations were sent to the medical profession throughout Marion and the adjoining counties.



Eye, Ear, Nose and Throat Operating Room.

Sterilizing Room.

and at 4:30 p. m. some forty of our good doctor friends were escorted through the building by the superintendent, Mr. John A. Bowman, and the medical staff of the hospital, after which they met at the Marion Hotel, where they were the guests of the Staff at a banquet. Many short talks were given by the visiting doctors and some of the Board of Trustees, all expressing their



General Operating Room.

approval of the well-equipped institution. Dr. R. B. McIver, assisted by Dr. William McL. Shaw, both of Jacksonville, entertained the entire company with his very interesting discussion of lesions of the genito-urinary tract with particular reference to kidney, illustrated by lantern slides.

A DISCUSSION OF THE CAUSE OF LABOR WITH REPORT OF A CASE WHERE LABOR FAILED TO DEVELOP*

W. B. Ryan, M.D.,
Coral Gables.

At the onset of labor the intermittent contractions of the uterus which have existed throughout pregnancy, merge into, or are replaced by contractions of a painful and more forcible nature. These contractions increase in strength and severity and result in dilatation of the cervix and expulsion of the child and placenta.

The cause of this process is unknown. Many theories have been adduced by various investigators but none seem capable of universal application. Some obstetricians believe that it is a simple development of the intermittent contractions of pregnancy augmented by some slight irritation either emotional or mechanical. In support of this they cite the known readiness with which the uterus reacts to stimulation in the latter months of pregnancy and the fact that the intermittent contractions are often painful for two or three weeks before labor.

Mauriceau and many others since his time believed that labor was entirely a reaction to distention, that when the uterus was distended to a certain point it reacted as any other hollow organ and attempted to empty itself. They cite as proofs the frequent occurrence of premature labor in the presence of hydramnios or twin pregnancy. On the other hand we often find premature labor with small children and prolonged pregnancy with large ones.

Galen's explanation, which still has many adherents, was that labor is due to pressure of the presenting part on the cervix. But pressure on the cervix with varying degrees of dilatation may exist for days without labor and labor begins in transverse presentations where no presenting part is in contact with the cervix.

Keilmann and Knüpffer brought forward the theory that labor was due to the formation of the lower uterine segment causing pressure on the surrounding nervous ganglia. Labor may begin with a rigid cervix before the lower uterine segment is formed.

Naegele, Simpson, Scanzoni and others believed that in the latter weeks of pregnancy the decidua underwent a fatty degeneration causing

*Read before Dade County Med. Soc., Aug. 6, 1927.

a partial separation of the ovum. The ovum then acted as a foreign body and the uterus contracted to expel it. Later investigations have shown that this change is not normal and does not occur in the great majority of pregnancies. The septa become thinner but are not torn through till the expulsion of the foetus.

Geyle and others believed that labor was a race habit of great antiquity. That nature after ages of experimentation in the dawn of man's existence found that two hundred and eighty days was the most favorable time for both mother and child. Longer pregnancies resulted in large children with difficult labors and lacerations while shorter pregnancies brought forth puny babies with a high death rate. This theory, however, will not legally account for the birth of children in Tennessee.

Undue exercise or exertion, jars, violence, strong emotion, grief or anger may and often do induce labor. These causes are not the usual or normal manner of terminating pregnancy and we do not know the exact mechanism by which they work.

Eden and Williams believed that labor might be due to senility of the placenta as evidenced by infarct formation. They thought that due to this senility the foetus may lack nutrition and this lack of nutrition give rise to metabolic waste products which enter the maternal circulation and stimulate the uterus to contract. Many placentae however show no infarcts, chalk, cysts, cicatrices or other evidence of senility.

Brown, Sequard and later Keiffer demonstrated that an excess of carbon dioxide in the blood would cause uterine contractions, but no one has proved the presence of an excess of carbon dioxide in the blood during or before labor. Williams showed that during labor the output of carbon dioxide is lowered. The great muscular exertion should produce an increase so there must be some bodily change that depresses the oxidative processes.

The investigations of Slemons showed marked changes in metabolism a few hours before labor. There was a profuse diuresis with lowered nitrogen output. This is opposite to the usual condition during the greater part of pregnancy. These changes were not present in cases of induced labor.

Mende, Tyler-Smith, Löwenhardt, Beard and others support the ovarian hormone theory. They believe that during pregnancy a hormone is formed by the ovary or the corpus luteum. This hormone is most plentiful at the time when the menses would be due and accounts for the increased tendency of the uterus to contract at those times. At the tenth menstrual cycle this hormone reaches its height and causes labor.

Von de Heide in his experiments found that the intravenous injection of foetal serum into pregnant women would in some cases cause labor. He argued from this that labor was an anaphylactic reaction. That foetal substances gained entry into the maternal circulation and caused the formation of antibodies. As term approached the foetal antigen became excessive and the reaction of antigen and antibodies set free substances that stimulated the uterine ganglia and produced labor.

Spiegelberg advocated a theory of foetal causation similar to that of Eden and Williams except for the factor of placental senility. He believed that the foetus reached a stage of development at which the placental circulation no longer furnished the full amount of materials needed for growth and further development. This malnutrition of the foetus resulted in foetal excreta entering the maternal circulation and caused labor.

Kieffer and others worked out the nerve supply of the uterus and proved it to be of threefold origin. Part is derived from the lumbar cord, part from the sympathetic and part from the intrinsic ganglia and nerves of the uterine muscle. Kruieger and Offergeld in their animal experiments cut the pregnant uterus free from all connection with the central and the sympathetic nervous system and found that labor set in at the usual time and progressed in a normal manner. This proved that labor is not dependent on any center in the central nervous system and they argued that it must be due to stimulation of the intrinsic uterine ganglia by unknown substance in the circulation.

Sauerbruck and Heyde, whose work was afterwards confirmed by Williams, united female rats in such a manner that they were living in symbiosis. If both rats were pregnant labor in one caused labor in the other. If only one rat were pregnant the onset of labor caused serious illness of the other. This would prove that labor is due to some substance circulating in the blood which is harmless to pregnant animals but poisonous to the non-pregnant. The nature of this substance and whether it is of maternal, foetal or placental origin is unknown.

There is probably a second factor which acts independently or in conjunction with this substance and is concerned in labor induced by emotional or mechanical irritation.

I wish to report a case in which for three successive pregnancies labor failed to set in at term.

Mrs. S. W. V., seen June 12, 1926, white, American of German ancestry, age twenty-seven; height five feet five inches; weight one hundred and forty-two pounds.

Last menstruation December 2, 1925; probable date of confinement September 15, 1926.

Family history negative. Personal history: Usual diseases of childhood. Thyroid gland began to enlarge at age of thirteen. Menstruated at fourteen. Thyroid continued to enlarge and at seventeen began to exhibit toxic symptoms but improved under X-ray therapy. She had influenza in 1918.

First pregnancy in 1921. Pregnancy progressed normally but labor did not occur at the expected time. Her physician thought that she had miscounted and allowed her to go another month. Still labor did not occur and the pressure symptoms becoming distressing he endeavored to induce labor with quinine and castor oil. He tried pituitrin. He inserted a bougie and packed the cervix and vagina with gauze. This failing he waited two weeks more and then removed her to a hospital and inserted a series of Voorhees bags. These failed to produce pains so he dilated the cervix manually, applied high forceps and delivered a large child with head marked by forceps. Child lived a few minutes.

Second pregnancy in 1923 with a different physician. Again she was allowed to go a month over term. Again castor oil, quinine, pituitrin, bougies, and bags were tried without success. She was delivered by manual dilatation and podalic version. This child lived.

Physical examination: Well nourished and developed; moderate enlargement of thyroid; slight exopthalmos-Bruit over thyroid; heart slightly irregular, no enlargement or murmurs; pulse 88; blood pressure 122-74.

Uterine fundus one and one-half inches above umbilicus; foetal heart not heard; foetal position not determined; pelvic measurements normal; vaginal outlet relaxed; old stellate laceration of cervix.

Pregnancy progressed normally; urine and blood pressure normal; pulse continued rapid and slightly irregular.

In August I discovered that she had a twin pregnancy. She passed the expected date of confinement but due to the damaging of her residence by the hurricane and the crowded conditions of the hospital I waited until the house could be repaired and the lights turned on again.

By October first she had had no pains and was greatly distended and very uncomfortable. Her legs were swollen and painful due to pressure on the vessels and nerves. On examination I found the cervix obliterated and two fingers dilated. The foetal head was engaged. I gave her two ounces of castor oil and followed it with four ergo apiol capsules and five grains of quinine every hour for six doses. No pains resulted.

The same dosage was repeated on October 2nd with the same result.

On October third I inserted two silk catheters between the membranes and the uterine wall and packed the cervix and vagina with gauze.

On October fourth, with catheters and packing still in position, I gave ergo apiol, quinine and pituitary extract every hour for six doses, starting with three minims of pituitary extract and increasing two minims each dose. I continued this on October fifth, using 1 c.c. of pituitary extract at a dose. I tried three different makes of pituitary extract—I never succeeded in producing a single pain but several times I was almost sure that I could feel a slight contraction of the uterus.

The patient and myself both rested on October sixth and the following day under general anesthesia I dilated the cervix by hand, ruptured the membranes, applied forceps and delivered a male child weighing six and one-half pounds. I then ruptured a second sac and delivered a female child, five and three-quarter pounds in weight, by pressure on the fundus. The placenta was expressed manually and the uterus contracted down firm and hard. The puerperium was normal and mother and children continue in good health.

I am unable to form any definite opinion as to the cause of her failure to go into labor but I suspect that her thyroid has something to do with it.

I have omitted the bibliography as no original observations are included and no claim for originality is made.

A NEW UTERINE DILATOR*

HAMILTON COOKE, M. D.
Fellow in Surgery,
The Mayo Foundation, Rochester, Minn.

Dilatation of the cervical canal may require considerable force. This force must be applied slowly and gently; it must also be under constant complete control in order not to rupture or even to injure the cervix.

A new dilator, using the principle of double leverage, has been designed to meet these requirements. The instrument consists of three parts, the blades, the levers, and the handles. The blades are 7 cm. long, with a gentle upward curve, and with smooth ridges on the lateral surface to prevent them from slipping out of the cervical canal. They diverge in a V-shaped manner affording, at complete dilatation, a spread of 4 cm. at the tip and 2 cm. at the base (Fig. 1). The second part is 9 c.m. long and consists of a double lever. The handles, which are curved downward, are 14 cm. long with grooves on the lateral surfaces. When the dilator is closed the handles are 12.5 cm. apart at the base (Fig. 2). The handles are sufficiently approximated before the initial resistance is encountered to fit comfortably in the average size contracting hand.

The principle of double leverage employed in this dilator gives a mechanical advantage of x5. That is, the force delivered by the dilating blades is five times that applied on the handles. This mechanical advantage is obtained by having the dilating blades open only one-fifth as rapidly as the handles close. The dilatation is thus not only powerful but also, as is essential, very slow. The powerful dilatation is an advantage to the operator in dilating the rigid resistant cervix of the nullipara and in cases of stricture and stenosis of the cervical canal. The slow dilatation is an important factor in dilatation of a soft edematous cervix, which may be easily injured or even ruptured by rigid uncontrolled force. The V-shaped separation of the blades affords the maximum of dilatory force at the site of greatest resistance, the external os and the region of the internal os.

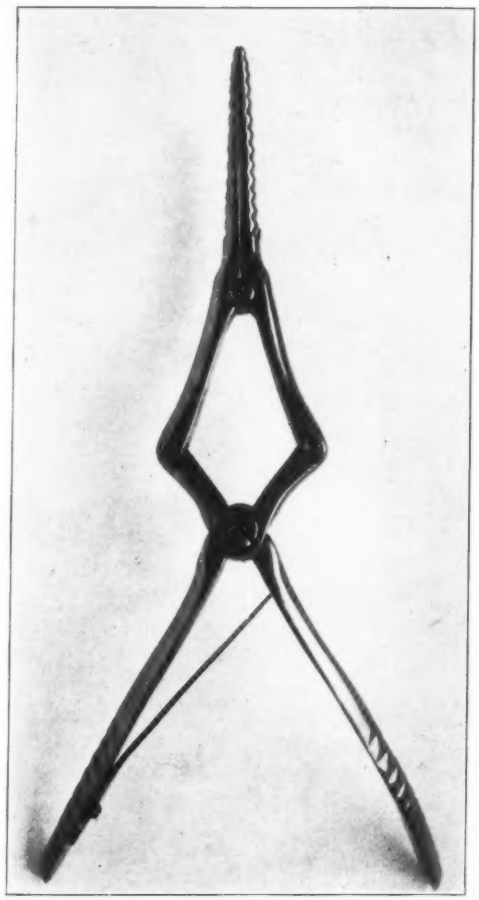


Figure 2.

The advantages of this instrument are: (1) simple construction, (2) slow dilatation, (3) powerful dilatation (mechanical advantage x5), (4) maximal force delivered at point of greatest resistance, and (5) unusually large force under constant control due to the slow dilatation.

*Work done in Section on Surgical Pathology, Mayo Clinic.

Figure 1.

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TUBERCULOSIS AND THE PERIODIC HEALTH EXAMINATION

The death rate from all forms of tuberculosis among the industrial populations of the United States and Canada, has declined nearly 40 per cent within seven years. The rate per annum among insured white persons for the first nine months of 1927 was 76.2 per 100,000, more than 8 per cent lower than any previous record. Better housing and ventilation, better food, more out-of-doors activity, pure air and sunshine, hospitalization of patients, fewer debilitating habits and hardships are doubtless factors contributing largely to this saving of human life, but the early recognition of activity, so essential to the successful "cure," may well be given a place of equal importance.

The periodic health examination, including careful history and inspection, palpation, percussion and auscultation, may be sufficient to exclude or detect early activity, but the roentgram will

often reveal lesions not detected by other means.

Justice to himself as well as to the applicant requires that all diligence be given the task if the periodic health examination or the physical examinaton of the chest is undertaken.

An early diagnosis campaign to be conducted throughout the month of March is being sponsored by the Florida State Board of Health, the Florida Public Health Association and all affiliated organizations in cooperation with the American Medical Association and national health agencies. This will bring to the medical profession many opportunities to render real professional service.

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Nabers, L. W., 3618 N. E. 2nd Ave. ..... Miami
Newell, C. E., 1207 W. Flagler St. ..... Miami
Newton, S. B., 168 Sea Breeze Ave. ..... Palm Beach
Nichol, E. S., 306 Huntington Bldg. ..... Miami
O'Quinn, L. H. ..... Hialeah
Panettiere, Cayetano, Aladdin Bldg. ..... Miami Beach
Paulk, Geo. A., 202 Venetian Bldg. ..... Miami
Palmer, B., Calumet Bldg. ..... Miami
Payne, J. W., 2682 S. W. 8th St., Miami
Payton, F. J., Allison Hospital . . . Miami
Pearson, Homer, 1207 W. Flagler St. ..... Miami
Pearson, Nelson T., 1st Natl. Bk. Bldg. ..... Miami
Pearson, R. J., 210 E. Flagler St. ..... Miami
Peters, E., 506 Olympia Bldg. . . . . Miami
Preston, R. W., 314 Calumet Bldg. ..... Miami
Quillian, W., Laidlaw-Southworth Bldg. ..... Coral Gables
Rentz, L. S. ..... Coconut Grove
Rentz, W. C., 1744 N. W. 36th St. ..... Miami
Roche, C. F., Aladdin Bldg., Miami Beach
Ryan, W. B., 206-7 Karp Bldg. . . . . Coral Gables
Sayles, C. F., 312 N. W. 3rd Ave. ..... Miami
Shaw, E. Clay, 508 Huntington Bldg. ..... Miami
Shisler, J. W., 205 Shoreland Arcade ..... Miami
Simpson, J. R., 1051 Seybold Bldg. ..... Miami
Sinclair, J. A. B., 306 N. E. 16th St. ..... Miami
Skaggs, P. T., 145 E. Flagler St. ..... Miami
Smith, C. K., Tatum Bldg. ..... Miami
Smith, J. A. ..... Homestead
Smith, J. W., 1760 N. W. 5th St. ..... Miami
Smith, M., 405-6 Huntington Bldg. ..... Miami
Snyder, J. W., 402 Huntington Bldg. ..... Miami

* Deceased.

Spiegel, A., 104 Renaurt Bldg. . . . . Coral Gables
Stewart, J. S., 1049 Seybold Bldg. ..... Miami
Stimson, Horace, 402 Huntington Bldg. ..... Miami
Strong, S. M., 108 Main St. ..... Flushing, N. Y.
Stuart, J. D., 1224 Altamari Ave. ..... Coral Gables
Summers, Albert T., 142 E. Flagler St. ..... Miami
Tallman, M. H., 312 Venetian Arcade ..... Miami
Thomas, E. C., 45 N. E. 5th St. . . Miami
Thomas, M. D., Box 237 ..... Miami
Thorne, J. I., Seybold Bldg. ..... Miami
Threatte, Bruce, Huntington Bldg. ..... Miami
Threlkeld, M. E., 109 N. E. 6th St. ..... Miami
Tower, J. B., 30 Krome Ave., Homestead
Tumlin, C. E., 203 Townley Bldg. ..... Miami
Turner, J. C., 212 E. Flagler St., Miami
Vinson, W. J., 403 Prof. Bldg. . . . Miami
Vogt, F. A., 207 Calumet Bldg. . . . Miami
Walters, A. L., 337 Lincoln Road ..... Miami Beach
Ward, Frank ..... Fitzgerald, Ga.
Watters, W. H., Boston-Miami Clinic ..... Coconut Grove
Weiland, A. H., Calumet Bldg. . . Miami
Welch, P. D., 316-20 Calumet Bldg. ..... Miami
Westermann, J. T., 4025 N. E. 2nd Ave. ..... Miami
Whitaker, Joel, 905 Congress Bldg. ..... Miami
White, D. W., 513 Miami Bk. & Trust Bldg. ..... Miami
Whitten, B. L., 1st Natl. Bank Bldg. ..... Miami
Wiggins, J. C., 3618 N. E. 2nd Ave. ..... Miami
Wilson, M. C., 606 Huntington Bldg. ..... Miami
Wood, A. W., 416 Security Bldg., Miami
Yarbrough, H. C., 812 Bayshore Blvd. ..... Miami
Youmans, C. P. ..... Crescent City
Youmans, I. C., Prof. Bldg. ..... Miami

DE SOTO-HARDEE-HIGHLANDS COUNTY MEDICAL SOCIETY

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Weems, H. V., V.-Pres., 22 Oak St. ..... Sebring
Chandler, I. W., Sec.-Treas., First Trust Bldg. ..... Avon Park
Aurin, E. C. ..... Ft. Ogden
Bevis, H. P. ..... Arcadia
Brown, L. V. L. ..... Sebring
Coker, J. S., DeSoto Natl. Bank Bldg. ..... Arcadia
Crum, M. L. ..... Arcadia
Garner, J. E. ..... Wauchula
Highsmith, G. F. ..... Arcadia
Hubert, M. A. ..... Avon Park
Kayton, M. C. ..... Wauchula
Kirkpatrick, C. H., Box 454 . . . . Arcadia
Mitchell, J. W. ..... Sebring
Poucher, A. A. ..... Wauchula
Pyatt, W. S. ..... Bowling Green
Spears, B. D. ..... Zolfo Springs
Touchton, W. C. ..... Avon Park
Witt, C. C. ..... Arcadia

DUVAL COUNTY MEDICAL SOCIETY

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Driskell, S. E., Vice-Pres., St. James Bldg. ..... Jacksonville
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Shaw, W. McL., Treasurer, St. James Bldg. ..... Jacksonville
Adams, G. E., 6620 Buffalo Ave. ..... Jacksonville
Adams, Thos. S., 5 Main St., Jacksonville
Adams, W. H., Prof. Bldg. . . Jacksonville
Alford, Neil, St. James Bldg. ..... Jacksonville
Arms, B. L., State Board of Health ..... Jacksonville
Bacon, H., 401 W. Duval St. ..... Jacksonville
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Barfield, F. G., St. James Bldg. ..... Jacksonville
Baumgartner, C. J., 406 Masonic Bldg. ..... Jacksonville
Bayless, W. C., 203 St. James Bldg. ..... Jacksonville
Beals, J. A., Riverside Hospital . . . . Jacksonville
Beckman, G. E., Professional Bldg. ..... Jacksonville
Berry, N. C., 2017 Main St. ..... Jacksonville
Black, J. B., St. James Bldg. ..... Jacksonville
Blackmar, R. W., 319-321 St. James Bldg. ..... Jacksonville
Blinn, T. A., St. James Bldg. ..... Jacksonville
Boone, J. L., 520 Professional Bldg. ..... Jacksonville
Bowen, F. J., St. James Bldg. ..... Jacksonville
Boyd, J. E., 342 St. James Bldg. ..... Jacksonville
Bransford, L. E., Professional Bldg. ..... Jacksonville
Brillhart, H. L., Graham Bldg. ..... Jacksonville
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Brinson, W. D. ..... Baldwin
Broadbent, O. P., St. James Bldg. ..... Jacksonville
Buckman, T. E., 2708 St. Johns Ave. ..... Jacksonville
Carefoot, E. I., Professional Bldg. ..... Jacksonville
Cason, T. Z., Riverside Hospital . . . . Jacksonville
Chapman, B. A., St. James Bldg. ..... Jacksonville
Chilli, Joseph L., Main and 8th Sts. ..... Jacksonville
Collins, C. C., St. James Bldg. ..... Jacksonville
Copeland, S. M., St. James Bldg. ..... Jacksonville
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Cunningham, L. W., St. James Bldg. ..... Jacksonville
Day, Gaston, St. James Bldg. ..... Jacksonville
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Dean, Russell, St. James Bldg. ..... Jacksonville
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Fisher, L. C. ..... Green Cove Springs
Fort, F. L., Prof. Bldg. . . . Jacksonville
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Gwinn, V. H., 1935 Riverside Ave. ..... Jacksonville
Harrell, D. E., St. James Bldg. ..... Jacksonville
Harris, H. H., St. James Bldg. ..... Jacksonville
*Harris, R. L., Professional Bldg. ..... Jacksonville
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Holden, G. R., Wade Bldg., 1022 Park St. ..... Jacksonville
Holloway, L. W., St. James Bldg. ..... Jacksonville
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Ives, H. A., 15 Forrest St. . . Jacksonville
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Jennings, C. L., Professional Bldg. ..... Jacksonville
Johnston, C. W., St. James Bldg. ..... Jacksonville
Jones, F. C., Graham Bldg., Jacksonville

Keisling, F. C., 315 Professional Bldg. ..... Jacksonville
Key, Foster P. ..... Green Cove Springs
Killinger, R. R., St. James Bldg. ..... Jacksonville
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Kirk, W. W., Prof. Bldg. ..... Jacksonville
Knauer, W. J., Buckman Bldg. ..... Jacksonville
Knight, A. C., Prof. Bldg., Jacksonville
Love, J. D., 2063 Oak St. .. Jacksonville
Mabry, C. B., St. James Bldg. ..... Jacksonville
McEuen, H. B., 320 Professional Bldg. ..... Jacksonville
McGinnis, R. H., 2063 Oak St. ..... Jacksonville
McIver, R. B., St. James Bldg. ..... Jacksonvillle
McKenzie, A. C., St. James Bldg. ..... Jacksonville
Manhoff, Ben, 712 Laura St. ..... Jacksonville
Manning, W. S., St. James Bldg. ..... Jacksonville
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Mitchell, G. M., 712 Laura St. ..... Jacksonville
Moe, L. N., 212 St. James Bldg. ..... Jacksonville
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Norwood, J. K., Beaver and Hogan Sts. ..... Jacksonville
Oetjen, G. F., St. James Bldg. ..... Jacksonville
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Pasco, J. D., Wade Bldg. ..... Jacksonvile
Peterson, C. A., St. James Bldg. ..... Jacksonville
Peyton, H. A., Riverside Hospital ..... Jacksonville
Pittman, J. H. ..... West Palm Beach
Proctor, H. L., 210 Prof. Bldg., ... Jacksonville
Ramage, Raymond B., 219-20 Prof. Bldg. ..... Jacksonville
Randolph, J. H., St. James Bldg., ... Jacksonville
Reaves, H. A., Prof. Bldg. .Jacksonville
Richards, F., Prof. Bldg. ..Jacksonville
Richardson, G. W., 343 St. James Bldg. ..... Jacksonville
Richardson, Shaler A., 111 W. Adams St. ..... Jacksonville
Rollins, C. D., 2162 Riverside Ave. ..... Jacksonville
Ross, W. E., St. James Bldg., ... Jacksonville
Sanderson, Raymond, 3325 Orange Park Road ..... Jacksonville
Sandusky, C. M., 28 W. Monroe St. ..... Jacksonville
Schnauss, W. R., 312 Hildebrant Bldg. ..... Jacksonville
Sellers, E. T., St. James Bldg. ..... Jacksonville
Sengstak, E. P. E., ... Mandarin
Simpson, J. K., 712 Laura St. ..... Jacksonville
Skipper, C. T., St. James Bldg. ..... Jacksonville
Smith, R. E., 112 Julia St. ..... Jacksonville
Stollenwerck, A. D., 25 W. Beaver St. ..... Jacksonville
Swift, Edwin C., 2005 Park St. ..... Jacksonville
Taylor, H. M., 111 W. Adams St. ..... Jacksonville
Teeter, E. H., St. James Bldg. ..... Jacksonville
Thomas, R. Y. H., 502-4-6 Lynch Bldg. ..... Jacksonville
Thompson, D. C., 2579 Herschell St. ..... Jacksonville
Thompson, T. C., 318 Hildebrandt Bldg. ..... Jacksonville
Tyler, L V., Atlantic Blvd. and King Rd. ..... So. Jacksonville
Upchurch, N. A., City Board of Health ..... Jacksonville
Van Schaick, H. D., St. James Bldg. ..... Jacksonville
Veal, E. W., 128 St. Johns Ave. ..... So. Jacksonville

*Deceased.

Waas, F. J., Prof. Bldg. ...Jacksonville
Washburn, Clayton D., St. James Bldg. ..... Jacksonville
West, C. Omer., 500-5 Prof. Bldg. ..... Jacksonville
Wilcox, C. R., 712 Laura St. ..... Jacksonville
Wilkinson, A. H., 313 Prof. Bldg. ..... Jacksonville
Wilson, A. K., St. James Bldg. ..... Jacksonville
Wilson, J. F., St. James Bldg. ..... Jacksonville
Woolsey, B. F., 319-321 St. James Bldg. ..... Jacksonville
Wynn, Robert S., 305 Consolidated Bldg. ..... Jacksonville

ESCAMBIA COUNTY MEDICAL SOCIETY

Turberville, J. S., President .... Century
Bickerstaff, J. H., Vice-President, Blount Bldg. ..... Pensacola
Hoffman, James M., Sec'y-Treas., Pensacola Hospital ..... Pensacola
Ames, A. M., 206 Blount Bldg. ..... Pensacola
Bell, J. D., Blount Bldg. ..... Pensacola
Bryans, H. L., 21½ E. Wright St. ..... Pensacola
Bryans, R. L., 23 E. Wright St. ..... Pensacola
Carter, John H., 312-14 Brent Bldg. ..... Pensacola
D'Alemberte, C. W., 311 Blount Bldg. ..... Pensacola
Dewberry, W. C., Theisen Bldg. ..... Pensacola
Fellows, J. H., Brent Bldg. ... Pensacola
Fraser, Donald S., State Board of Health Lab. ..... Pensacola
Gachet, N. L. ..... Century
Haisfield, A. R., Blount Bldg. . Pensacola
Heinberg, C. J. ..... Pensacola
Hixon, F. P. ..... Pensacola
Holly, J. C. ..... Milton
Hutchinson, C. E., 303 Blount Bldg. ..... Pensacola
Johnson, J. R., Blount Bldg., Pensacola
Lischkoff, M. A., Blount Bldg., Pensacola
McClure, H. A. ..... Munson
Mixon, J. A., Box 697 ..... Pensacola
Nobles, R. G., Blount Bldg. ... Pensacola
Nobles, V. R., Blount Bldg. ... Pensacola
Nobles, W. D. ..... Pensacola
Payne, W. C., Blount Bldg. ... Pensacola
Peel, George T., Brent Bldg. .. Pensacola
Pierpont, J. H., American Bank Bldg. ..... Pensacola
Pollock, W. A. J., Jr., 303 Theisen Bldg. ..... Pensacola
Quina, M. E. ..... Pensacola
Renshaw, F. G., 104 S. Palafox St. ..... Pensacola
Simpson, H. L., 303 Theisen Bldg. ..... Pensacola
Snyder, H. D., Box 537, Knoxville, Tenn.
Stokes, T. H., Theisen Bldg. .. Pensacola
Sullivay, R. L., Route 1, Box 90 ..... Pensacola
Thames, Rufus ..... Milton
Turner, J. B. ..... Bagdad
Webb, C. C., 303 Blount Bldg., Pensacola

HAMILTON COUNTY MEDICAL SOCIETY

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Corbett, J. H., Vice-Pres. ..... Jasper
Barnett, R. A., Sec'y-Treas. ..... White Springs
Beaty, Jas. J. ..... Jennings
Box, C. C. ..... Willow
Bruce, John Robert ..... Jasper

HILLSBORO COUNTY MEDICAL SOCIETY

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Martin, D. D., Vice-Pres., 906 So. Rome Ave. ..... Tampa
Lowry, B. W., Sec'y-Treas., Citrus Ex. Bldg. ..... Tampa
Adamo, F. S., 3012 7th Ave., Ybor City ..... Tampa
Adamson, W. P., 766 Franklin St. ..... Tampa
Allen, Bundy, Citizens Bk. Bldg. ..... Tampa
Alsobrook, J. W. ..... Plant City
Andrews, C. A., Citizens Bank Bldg. ..... Tampa
Arroyo, C. F., 1425½ E. Broadway ..... Tampa
Baldwin, R. E., Citizens Bk. Bldg. ..... Tampa
Barker, F. T., 302 Krause Bldg. ..... Tampa
Bartlett, C. W., 1521 7th Ave. ..... Tampa
Beyer, A. R., Box 474 ..... Tampa
Bidwell, A. M., 411 1st Natl. Bk. Bldg. ..... Tampa
Bitzer, E. W., Citizens Bank Bldg. ..... Tampa
Black, R. C., 101 San Ever St. ..... Tampa
Blake, W. C., Citz. Bank Bldg. ..... Tampa
Blackman, H. J., Citz. Bank Bldg. ..... Tampa
Bottari, G. C., 1820½ 7th Ave. ..... Tampa
Brown, G. W., Jr., Citizens Bank Bldg. ..... Tampa
Butchard, J. R., 112 N. Boulevard ..... Tampa
Cameron, F. A., Gas Bldg. ..... Tampa
Carlton, L. F., Citizens Bk. Bldg. ..... Tampa
Carter, E. F., 1719 Grand Central Ave. ..... Tampa
Chandler, J. C., Citrus Exch. Bldg. ..... Tampa
Christian, Geo. R., 200-1 Krause Bldg. ..... Tampa
Clark, W. F., 304 Krause Bldg. ..... Tampa
Cook, George L., 906 So. Rome Ave. ..... Tampa
Cowart, J. T., 906 So. Rome Ave. ..... Tampa
Crum, J. W., 2023½ 7th Ave. ..... Tampa
Daniels, B. A., 608 Tampa St. ..... Tampa
Dickinson, J. C., 302 Citz. Bk. Bldg. ..... Tampa
Draper, A. D., 5607 Florida Ave. ..... Tampa
Duke, R. R., 708 Citz. Bank Bldg. ..... Tampa
Duncan, W. P., 201 Krause Bldg. ..... Tampa
Dyer, W. H., 2201½ 7th Ave. ..... Tampa
Edwards, W. E., ... Safety Harbor,
Efrid, L. J., 509 Stovall Bldg. ... Tampa
Ely, R. A., 404½ Zack St. ..... Tampa
Estes, J. L., Stovall Bldg. ..... Tampa
Farrior, J. B., Citz. Bk. Bldg. . Tampa
Favor, H. M., 402 Citrus Exch. Bldg. ..... Tampa
Fluker, C. B., Box 105 ..... Tampa
Forbes, S. B., Citz. Bk. Bldg. ... Tampa
Foster, J. C., 1801½ 21st St. ... Tampa
Gale, John S., 5403 Florida Ave. ..... Tampa
Gilbert, Elsie, 6508 Central Avenue ..... Tampa
Gilmer, E. S., Citizens Bank Bldg. ..... Tampa
Glass, R. E., 914 14th Ave. ..... Tampa
Grantham, J. M., 436 W. Lafayette St. ..... Tampa
Gyland, Stephen P., Stovall Bldg. ..... Tampa
Hamner, Geo. P., Citizens Bank Bldg. ..... Tampa
Hampton, H., P. O. Box 2113 ..... Tampa
Hardy, G. E. W., 818 First National Bank Bldg. ..... Tampa
Helms, J. S., Citizens Bank Bldg. ..... Tampa
Henderson, R. P., 613 Citizens Bank Bldg. ..... Tampa
Higgins, A. F., Lafayette Arcade Bldg. ..... Tampa
Holloway, E. W., 2023½ E. Broadway ..... Tampa
Holton, W. J., 119 E. Reynolds St. ..... Plant City
Hopkins, C. D., City Health Dept. ..... Tampa
Hopkins, W. B., Citrus Exchange Bldg. ..... Tampa
Hubbard, R. C., 2220 7th Ave. .. Tampa
Ives, A. C., Hinson Bldg. ..... Tampa
Jefferson, R., 818 1st National Bank Bldg. ..... Tampa
Jenson, H. J., 7303 Nebraska Ave. ..... Tampa
Jobson, A. M. C., 803 Citizens Bank Bldg. ..... Tampa
Jones, A. B., Jr., Bayside Hospital ..... Tampa
Knauf, A. R., 812 Citizens Bank Bldg. ..... Tampa
Knight, J. C. ..... Plant City
Lake, E. T., 8201 10th St. ..... Sulphur Springs

Lancaster, W. J., 325 Lafayette Arcade Bldg. ..... Tampa
Levy, E. C., City Health Officer ..... Tampa
McCaslan, W. H., 610 Citizens Bk. Bldg. ..... Tampa
McEachern, J. R., 205 Zack St. ..... Tampa
McMurray, H. E., 203 Memorial Highway ..... Tampa
McRae, E. H., Citrus Exch. Bldg. ..... Tampa
Maechtle, E. W., 2609 Sunset Drive ..... Tampa
Maguire, T. C., ... Plant City
Meighen, Douglass, Gas Bldg.. Tampa
Mills, David, 2107 N. Jefferson.. Tampa
Mills, J. H., 910 E. Michigan Ave. ..... Tampa
Mitchell, L. B., Box 737 ..... Tampa
Moore, J. T., Tribune Bldg. ... Tampa
Nelken, B. E., 7303 Nebraska Ave. ..... Tampa
Nelson, R. G., 712 Citizens Bank Bldg. ..... Tampa
Oglesby, C. R., P. O. Drawer E., ... Tampashores
Oppenheimer, L. S., 408 Citz. Bank Bldg. ..... Tampa
Ortega, Rafael, 1805 15th St. ..... Tampa
Pate, J. C., 1107-9 1st Natl. Bk. Bldg. ..... Tampa
Patterson, William, Citrus Exch. Bldg. ..... Tampa
Rankin, G. C., Box 2233 Ave. ..... Tampa
Rector, L. T., 1210 E. Powahatan ..... Tampa
Rowlett, W. M., Citizens Bank Bldg. ..... Tampa
Rudisill, C. A., First Natl. Bank Bldg. ..... Tampa
Sexton, J. J., 205 Zack St. .. Tampa
Shaver, E. F., 2207½ 7th Ave. ..... Tampa
Smith, Burdette, Citrus Exchange Bank Bldg. ..... Tampa
Smith, H. Mason, 903 Theater Bldg. ..... Tampa
Smith, W. H. Y., City Health Dept. ..... Tampa
Smoak, Edw., 315 Citizens Bank Bldg. ..... Tampa
Snow, H. O., 1719 Grand Central Ave. ..... Tampa
Spengler, N. L., 903 Tampa Theater Bldg. ..... Tampa
Stringer, Sheldon, P. O. Box 162 ..... Tampa
Taylor, J. W., 807 Citizens Bank Bldg. ..... Tampa
Thompson, H. O., 5309 Central Ave. ..... Tampa
Thorpe, Franklin, 201-3 Lafayette Arcade ..... Tampa
Torbet, R. F., 819 Citizens Bank Bldg. ..... Tampa
Truelson, Thomas, Citizens Bank Bldg. ..... Tampa
Vinson, J. C., Box 571 ..... Tampa
Weathersbee, A. B., Lafayette Arcade Bldg. ..... Tampa
Weekly, A. S., Lafayette Arcade Bldg. ..... Tampa
Williams, F. E., 1210 E. Powahatan ..... Tampa
Williams, Horace, 436 W. Lafayette St. ..... Tampa
Williams, J. W., Lafayette Arcade Bldg. ..... Tampa
Wilson, S. S., 618 Citizens Bank Bldg. ..... Tampa
Winton, M. R., Citrus Exchange Bldg. ..... Tampa
Yager, R. E., 436 W. Lafayette, Tampa

INDIVIDUALS

Green, O. F. ..... Mayo
Paul, L. H. ..... Bonifay

JACKSON COUNTY MEDICAL SOCIETY

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Hudgens, Thomas H., Sec'y-Treas. ..... Sneads
Baltzell, N. A. ..... Marianna
Boothe, W. R. ..... Campbellton
Burns, M. Q. ..... Blountstown
Dowling, J. B. ..... Alliance
Finlay, D. H. ..... Blountstown
Harper, C. W. ..... Chipley
Harrison, C. H. ..... Cottondale
Hodges, G. S. ..... Marianna
Kennedy, R. L. ..... Malone

* Deceased.

McLeod, J. E. ..... Cypress
Miller, R. L. ..... Graceville
O'Hara, A. M. ..... Cypress
Price, C. J. ..... Alford
Ryals, C. H. ..... Grand Ridge

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Ashton, W. Lee ..... Umatilla
Brady, Clyde ..... Leesburg
Calvin, Wm. J., Palm Pharmacy Bldg. ..... Eustis
Coupland, J. D. ..... Eustis
DeVane, W. C. ..... Groveland
Fenn, H. T. ..... Mt. Dora
Hannum, M. M. ..... Eustis
Hawkins, A. S., Rt. 5, Box 116 B, ... Muskogee, Okla.
Holland, H. G., 202 State Bank Bldg. ..... Leesburg
Lodor, Chas. H., Palm Pharmacy Bldg. ..... Eustis
Lott, Walter M. ..... Clermont
Toy, S. H. ..... Umatilla
Tyre, C. McK. ..... Eustis
Williams, D. B. ..... Mt. Dora

LEE COUNTY MEDICAL SOCIETY

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Grace, W. H., Sec'y-Treas., Earnhardt Bldg. ..... Ft. Myers
Anderson, J. M. ..... Sears
Harrison, W. A. ..... Ft. Myers
Hunter, A. P., Langford Bldg., Ft. Myers
Johnson, M. F. ..... Ft. Myers
Jones, H. Quillian, 9 Leon-Sims Bldg. ..... Ft. Myers
Longbrake, G. A., 897 2nd St., Ft. Myers
Martin, Leldon W., P. O. Box 686, ... Punta Gorda
Merrick, Chas. G., Pattio de Leon, ... Ft. Myers
Nowling, J. C., 11 Theatre Arcade ..... Ft. Myers
Parnell, H. E., Langford Bldg., ... Ft. Myers
Seebold, J. L. ..... LaBelle
Stone, Geo. S., 2nd and Hendry, ... Ft. Myers
Whisnant, B., 3 Leon Bldg... Ft. Myers

LEON-GADSDEN-LIBERTY-WAKULLA-JEFFERSON COUNTY MEDICAL SOCIETY

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Brevard, E. M. ..... Tallahassee
Brinson, J. B., Jr., Dogwood St. ..... Monticello
Center, A. E. ..... Apalachicola
Davis, J. C., Jr., 203-8 Masonic Temple ..... Quincy
Dozier, L. L., St. Joseph's Hospital ..... Savannah, Ga.
Folmar, J. Q., Florida State Hospital ..... Chattahoochee
Glover, J. B. ..... Monticello
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Gwynn, G. H., Sr. ..... Tallahassee
Johnston, J. K., 111½ S. Monroe St. ..... Tallahassee
Kendrick, O. G. ..... Tallahassee
Murrow, J. S. ..... Apalachicola
Palmer, H. E. ..... Tallahassee
Robertson, M. C., College Infirmary F. S. C. W. ..... Tallahassee
Rhodes, B. M. ..... Tallahassee
Walker, Wm. H. ..... Lamont
Weems, Geo. E. ..... Apalachicola
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Wilkinson, B. A., Telephone Bldg. ..... Tallahassee
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Blake, L. W. ..... Bradenton
Brown, J. O. ..... Palmetto
Bryan, C. H. ..... Bradenton
Byrd, Hiram, 310 Citrus Exchange Bldg. ..... Tampa
Cowell, E. H., Children's Clinic, ... Bradenton
Fields, C. H., Park St..... Bradenton
Gates, H. ..... Bradenton
Harrison, M. M. ..... Palmetto
Haygood, J. K., 518 15th St.. Bradenton
Hollingsworth, S. G., 451 12th St. ..... Bradenton
Lancaster, B. M. ..... Manatee
Larrabee, C. W., Larrabee's Hospital, ... Bradenton
Leffingwell, J. B., 1st National Bank Bldg. ..... Bradenton
Luke, J. J. ..... Ellenton
McCoy, Thomas ..... Bradenton
McDuffie, T. M. ..... Manatee
*Markey, M. R. ..... Parish
Mason, J. F. ..... Bradenton
Overstreet, Ed. J. ..... Bradenton
Sporeman, M. P. ..... Manatee
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MARION COUNTY MEDICAL SOCIETY

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Chalker, J. L., Sec'y-Treas., 108 S. Magnolia St..... Ocala
Baskin, J. G. ..... Dunnellon
Dozier, H. C., 9 Magnolia St..... Ocala
Gatrell, Henry ..... Fairfield
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Lindner, E. G. ..... Ocala
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Moore, J. N. ..... Ocala
Slaughter, T. K. ..... Oxford
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Von Engleken, L. H. ..... Ocala
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Pintado, N. C. ..... Key West
Porter, J. Y., Jr., 417 Eaton St., ... Key West
*Porter, J. Y., Sr..... Key West
Warren, Wm. R., 511 Eaton St., ... Key West

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Andrews, Mitchell M., Vice-Pres., Clinic Bldg. ..... Orlando
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Anderson, Claude ..... Holopaw
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Balkins, A. J., Washington Sanatorium..... Takoma Park, Md.
Beardall, H. M. ..... Orlando
Brinson, H. ..... Kissimmee
Burks, B. Auxford, 108 E. Park Ave. ..... Winter Park
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Coffin, C. E. ..... Winter Park
Collins, Charles J., 11 Lucerne Circle ..... Orlando
Copp, F. A., 458 St. James Bldg. ..... Jacksonville
Davila, J. A., 25 E. Central Ave. ..... Orlando
Day, Horace A., Clinic Bldg., Orlando
Dodds, Wm. H. ..... St. Cloud
Edwards, Gaston H., Clinic Bldg. ..... Orlando

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Folsom, Spencer A., 11 Lucerne Circle ..... Orlando
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Gardner, J. F. ..... Winter Park
Geiger, H. S. ..... Kissimmee
Gray, Frank D., 11 Lucerne Circle ..... Orlando
Gwynn, H. W. ..... Orlando
Harms, F. H., 40 N. Orange St. ..... Orlando
Hoffman, C. D., Phillips Bldg., Orlando
Hotard, Roland F., 226 E. Park Ave. ..... Winter Park
Ingram, L. C. ..... Orlando
Johnston, Hewitt ..... Orlando
Kime, R. R. ..... Orlando
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Marshall, C. J. ..... Sanford
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Osenbach, William, 413-14 State Bank Bldg. ..... Orlando
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Perkins, Herman ..... Holopaw
Person, W. C., P. O. Box 571, Orlando
Pines, John A., Orlando Clinic, Orlando
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Rivers, Thomas M. ..... Kissimmee
Scott, Sam. R. ..... Ocoee
Shellhouse, L. H. ..... Ocoee
Sinclair, K. Y., 33 Aylmer Ave., ... Ontario, Canada
Sinclair, W. E., Clinic Bldg., Orlando
Spiers, Wm. H., 209 Clinic Bldg., ... Orlando.
*Toles, L. W. ..... Orlando

PALM BEACH COUNTY MEDICAL SOCIETY

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Stone, Vale D., Vice-Pres., 411 Comeau Bldg...West Palm Beach
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Heath, G. W., Treas., Commercial Bank Bldg..... West Palm Beach
Arnold, Wilbur O., 118 Myrtle St.... West Palm Beach
Baldwin, R. H., Box 3493 ..... West Palm Beach
Bazemore, Mary K., 119 S. Narcissus St..... West Palm Beach
Binkley, John F., 1206 Harvey Bldg. ..... West Palm Beach
Blair, W. M., Room 11, McGinley Bldg. ..... West Palm Beach
Brantley, Grady H., 811½ Lake Ave..... Lake Worth
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Buck, W. J., 1307 Harvey Bldg. ..... West Palm Beach
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Gerlach, Earl B., 523 Comeau Bldg. ..... West Palm Beach
Gill, Richard S., P. O. Box 1105, ... West Palm Beach
Gunter, T. D., Guaranty Bldg. ..... West Palm Beach
Hall, John E., Harvey Bldg., ... West Palm Beach
Henry, G. F., Citizens Bank Bldg. ..... West Palm Beach
Herman, F. Peter, 1007 Harvey Bldg. ..... West Palm Beach
Herpel, Frederick K., Good Samaritan Hosp. ..... West Palm Beach

*Deceased.

Jared, Vernon M., State Laboratory Bldg. ..... West Palm Beach
Lewis, R. G. ..... West Palm Beach
Merrill, C. M. ..... Palm Beach
Miller, Alice R..... West Palm Beach
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Smart, B. F. ..... Lake Worth
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Tatum, P. A. ..... West Palm Beach
Van Landingham, Wm. E., Citizens Bank Bldg..... West Palm Beach
Webb, Roy ..... West Palm Beach
Weems, Nat. M. ..... Boynton
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Cross, Kilpatrick ..... Inverness
Hancock, Wm. S., Jr., Murphy Bldg. ..... Brooksville
Hudson, P. J. ..... Crystal River
MacGregor, Geo. G. ..... Dade City
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Moon, Wm. B. ..... Crystal River
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Albaugh, Andrew P..... Tarpon Springs
Anderson, Wm. D., Box 53..... Largo
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Black, M. E. ..... Clearwater
Brown, Harold O., Coachman Bldg. ..... Clearwater
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Burnette, Elmer W., 1st Nat. Bank Bldg. ..... Tarpon Springs
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Cooper, J. H., 1st Nat. Bank Bldg. ..... St. Petersburg
Cranford, J. F., 736 Central Ave. ..... St. Petersburg
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Davis, W. M., 342 1st Ave., N., ... St. Petersburg
Dawson, S. A., Suite 105-107 Medical Arts Bldg. ..... St. Petersburg
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Echard, T. B., 1st Nat. Bank Bldg. ..... St. Petersburg
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Funk, Neil E., 1st Natl. Bank Bldg. ..... St. Petersburg
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Hooper, C. A., 419 2nd Ave., No., ... St. Petersburg
Hudson, Arthur T., 212 1st Nat. Bank Bldg. ..... St. Petersburg
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Knowlton, R. H., Power & Light Bldg. ..... St. Petersburg
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Lambdin, L. ..... St. Petersburg
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MacCordy, Earl C., Medical Arts Bldg. ..... St. Petersburg
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Miller, Geo. E., 208-210 Medical Arts Bldg. ..... St. Petersburg
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Moeller, Maximilian W., 525 1st Ave., N. ..... St. Petersburg
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Nettles, Robbins ..... Clearwater
Nickle, Millen A., 503-5 Coachman Bldg. ..... Clearwater
O'Brien, Raymond K., 105 5th Ave., No. ..... St. Petersburg
Osgood, G. E., Big Bayou, ... St. Petersburg
Peabody, J. D., 310 5th Ave., No., ... St. Petersburg
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Prather, B. T., 1st Natl. Bank Bldg., ... St. Petersburg
Quicksall, W. E., 222 Taylor Arcade, ... St. Petersburg
Remington, A. C., 304 West Ave., ... Rochester, N. Y.
Rieger, O. P., 1st Nat. Bank Bldg. ..... St. Petersburg
Roope, A. P., 815 Power & Light Bldg. ..... St. Petersburg
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Ruff, Joseph F., Bank of Clearwater Bldg. ..... Clearwater
Sackett, Harry R., 1027 15th Ave., No., ... St. Petersburg
Simcox, Lawrence, 213 3rd Ave., No., ... St. Petersburg
Solomon, H. D., Power & Light Bldg., ... St. Petersburg
Strickland, Jesse A., 712-14 Fla. Power Corporation ..... St. Petersburg
Stuart, M. H., 814 1st Nat. Bank Bldg. ..... St. Petersburg
Thomason, H. I., 6 Poinsettia Arcade ..... St. Petersburg
Timberlake, Gideon, 6th floor, Times Bldg. ..... St. Petersburg
Townsend, Shell H., Power & Light Bldg. ..... St. Petersburg
Welch, H. C., St. Petersburg Hall Bldg. ..... St. Petersburg
White, Benj. Lewis, 1st Nat. Bank Bldg. ..... St. Petersburg
Whitford, Grace R. ..... Ozona
Williams, Carl A., Power & Light Bldg. ..... St. Petersburg
Winchester, Harold E., Box 448, Dunedin
Wood, Alvin J., 801 1st Nat. Bank Bldg. ..... St. Petersburg

POLK COUNTY MEDICAL SOCIETY

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Biddle, P. D. .....Brewster
Carefoot, G. H. .....Ft. Meade
Causey, T. W., Box 1725.....Lakeland
Clark, S. A., 802 Marble Arcade Bldg. .....Lakeland
Cline, R. L. .....Lakeland
Clyatt, O. S., 908 So. Fla. Ave., ... Lakeland
Cordes, H. B. .....Frostproof
Deal, C. C., 208 Bank Bldg, Auburndale
Dickinson, W. P., 1003 Marble Arcade Bldg. .....Lakeland
Epling, B. D. .....Lake Wales
Farmer, C. H., City Hall.....Lakeland Bldg. .....Lakeland
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Gilbert, R. E. .....Winter Haven
Griffin, J. D., Polk Co. Trust Co. .....Lakeland
Hargrove, J. L., Polk County Hosp. .....Bartow
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Irons, F. E. .....Winter Haven
Justice, R. L. .....Haines City
Koon, A. C., 513 W. Lemon St., Lakeland
Leffers, Richard .....Lakeland
Lester, J. G., Marble Arcade, Lakeland
Lindsey, S. A. .....Ft. Meade
Love, C. W., Marble Arcade...Lakeland
McMurray, E. R. .....Bartow
Martin, E. E., 152 7th St., Haines City
Maynard, D. H. .....Lakeland
Murphy, H. K., Polk & Main Sts., ... Mulberry
Newman, H. P. .....Bartow
O'Cain, W. M. .....Bartow
Oglesby, J. M. .....Bartow
Overstreet, G. C., Marble Arcade Bldg. .....Lakeland
Pearce, C. C. .....Mulberry
Pennington, B. Y. .....Lake Wales
*Pennington, J. L. .....Lake Wales
Pillow, Robt., Jr., Pine Breeze Hospital .....Chattanooga, Tenn.
Ragsdale, V. H. .....Pierce
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Roberts, T. H., 716 N. Fla. Ave., ... Lakeland
Rogers, D. H., Jr. .....Haines City
Shafer, W. W. .....Haines City
Sherman, W. E., 716 W. Central Ave., ... Winter Haven
Simmons, T. G. .....Auburndale
Simpson, W. T. .....Winter Haven
Smith, S. F., Box 628.....Lakeland
Stetson, A. G. C., 941 S. Success Ave. .....Lakeland
Sullivan, R. R., 1006 Marble Arcade Bldg. .....Lakeland
Tillis, W. L., 215 Marble Arcade Bldg. .....Lakeland
Tomlinson, J. P. .....Lake Wales
Vassar, T. D., Strand Bldg., N. Ky. Ave. .....Lakeland
Weed, Walter A. .....Lakeland
Wilhoyte, R. E. .....Lake Wales
Williams, E. L. .....Ft. Meade
Wilson, C. H. .....Bartow
Wilson, J. F., 207-9 Spencer Futch Bldg. .....Lakeland

‡ PUTNAM COUNTY MEDICAL SOCIETY

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Rosborough, D. Y. .....Palatka
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Scruggs, S. A., Treas.....St. Augustine
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Guy, W. B., 52 Central Ave., ... St. Augustine
Hay, I. M. .....St. Augustine
Irwin, J. M. .....St. Augustine
Parkinson, W. N., East Coast Hospital .....St. Augustine
Potter, G. W., 83 King St., ... St. Augustine
Spencer, J. J. .....St. Augustine
Stanton, G. J. .....Hastings
Stevens, E. L., 45 Orange St., ... St. Augustine
Underwood, A. W., 48 Water St., ... St. Augustine
Webb, Walter D. .....St. Augustine
White, H. E., 401-3-5 First Nat. Bank Bldg. .....St. Augustine
Worley, S. G., 43 Jefferson Theatre Bldg. .....St. Augustine
Walton, Milton .....Hastings

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Boothe, R. C. .....Ft. Pierce
Clark, H. D. .....Ft. Pierce
Council, M. D., Arcade Bldg., Ft. Pierce
Davis, C. L. .....Okeechobee
Dunn, J. C. .....Ft. Pierce
Eurit, F. B. .....Stuart
Glidden, C. H. .....Ft. Pierce
Grossman, Frederick A. ....Vero Beach
Hardee, E. B. .....Vero Beach
Lingo, M. J. .....Okeechobee
Newnham, J. A. .....Stuart
Parker, J. D. .....Stuart
Whiddon, L. L. .....Ft. Pierce

SARASOTA COUNTY MEDICAL SOCIETY

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Metzger, F. C., Sec'y-Treas., Walpole Bldg. .....Sarasota
Cribbins, O. H., Bank of Sarasota Bldg. .....Sarasota
Halton, Jack, Miramar Court, Sarasota
Halton, Jos., Pineapple Ave., Sarasota
Harris, J. E., Bank of Sarasota Bldg. .....Sarasota
Johnston, W. J., Downey Bldg., Sarasota
Kennedy, David R., 1st Bank & Trust Bldg. .....Sarasota
Myers, N. P. .....Mayo
Nash, H. C., Downey Bldg.....Sarasota
Patterson, J. C., 1st Bank & Trust Co. .....Sarasota
Slocumb, C. B., Amer. Natl. Bank Bldg. .....Sarasota
Taylor, T. W., Walpole Bldg., Main St. .....Sarasota
Wilson, C. B., 1st Trust & Bank Bldg. .....Sarasota

SEMINOLE COUNTY MEDICAL SOCIETY

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Park, Chas. Lanier, Sec'y-Treas. ..... Sanford
Denton, J. F. .....Sanford
Langley, W. T., Meisch Bldg., Sanford
Mitchell, C. M. .....Sanford
Stevens, R. E., 1st and Palm Ave., ... Sanford
Tolar, J. N., 1st St.....Sanford

SUMTER COUNTY MEDICAL SOCIETY

Mitchell, W. E., Sec'y-Treas., P. O. Box 237.....Coleman
Albritton, A. B. .....Wildwood
Cherry, H. S. .....Center Hill
Clark, K. C. .....Bushnell
Wood, S. C. .....Leesburg

SUWANNEE COUNTY MEDICAL SOCIETY

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White, W. C., Sec'y-Treas.....Live Oak
Airth, H. F., 106 W. Howard St., ... Live Oak
Cline, D. E. .....Wellborn
Dicks, R. E. .....Dowling Park
Price, J. M. .....Live Oak

TAYLOR COUNTY MEDICAL SOCIETY

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Tyson, W. W., Vice-Pres.....Perry
Greene, Ralph J., Sec'y-Treas.....Perry
Bryan, W. H. .....Scanlon
Ellis, J. C. .....Perry
Richardson, John R. .....Carbur

VOLUSIA COUNTY MEDICAL SOCIETY

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Bates, Geo. L., 504½ Main St. Penn. Station .....Daytona Beach
Bohannon, Clyde C., 154 First Ave.....Daytona Beach
Bouchelle, Louis B. .....New Smyrna
Carter, E. A. .....DeLand
Carter, L. A. .....Bunnell
Chandler, J. R., 120 Volusia Ave. .....Daytona Beach
Chowning, W. C., 111 Palmetto St. .....New Smyrna
Clemmer, Chas. A., 913 Main St., ... Daytona Beach
Davis, C. W., 231 Coates St., ... Daytona Beach
Davis, Geo. A. .....DeLand
Davis, Joseph B. .....Daytona Beach
Dillard, T. H. .....DeLand
DuBois, Henry K.....Port Orange
Esche, J. P., 315 So. Penn. Drive .....Daytona Beach
Farmer, Myron H., 221 Orange Ave. .....Daytona Beach
Farnell, W. W. .....New Smyrna
Genge, V. P., 106 N. Ridgewood Ave. .....Daytona Beach
Heck, Maurice E., 223 Dreka Bldg. .....DeLand
Howe, Raymond, 229 N. Ridgewood Ave. .....Daytona Beach
Howe, Roy, 222 Volusia Ave. .....Daytona Beach
Johnston, Joseph L., 135 Canal St., ... New Smyrna
Merryday, H. L. .....Daytona Beach
Munson, Albert S., 310 Dreka Bldg. .....DeLand
Pay, W. C., 221 W. Rich Ave., DeLand
Rawlings, J. E. .....Daytona Beach
Smith, Dean T. .....Daytona Beach
*Stephens, J. W. .....DeLand
Taylor, Joseph E. .....DeLand
Taylor, Wm. H., 118 S. Beach St., ... Daytona Beach
Walter, F. J. .....LeMesa, Cal.
Weis, Edmund W. .....DeLand
West, Hugh .....DeLand
White, J. Blake .....Ormond Beach
Williams, W. J. .....Seville

WALTON-OKALOOSA COUNTY MEDICAL SOCIETY

Webb, E. Porter, Pres., City Pharmacy. ..... Crestview
Thorpe, E. D., Vice-Pres., ... DeFuniak Springs
Williams, A. G., Sec'y-Treas., Lakewood
Huggins, E. L. .....Freeport
McGuire, J. J. .....DeFuniak Springs
McSween, J. C. .....DeFuniak Springs
*Simmons, D. H. ....DeFuniak Springs
Spires, G. W. .....Darlington
Stephens, S. E. .....Laurel Hill

‡ Charter pending.
* Deceased.

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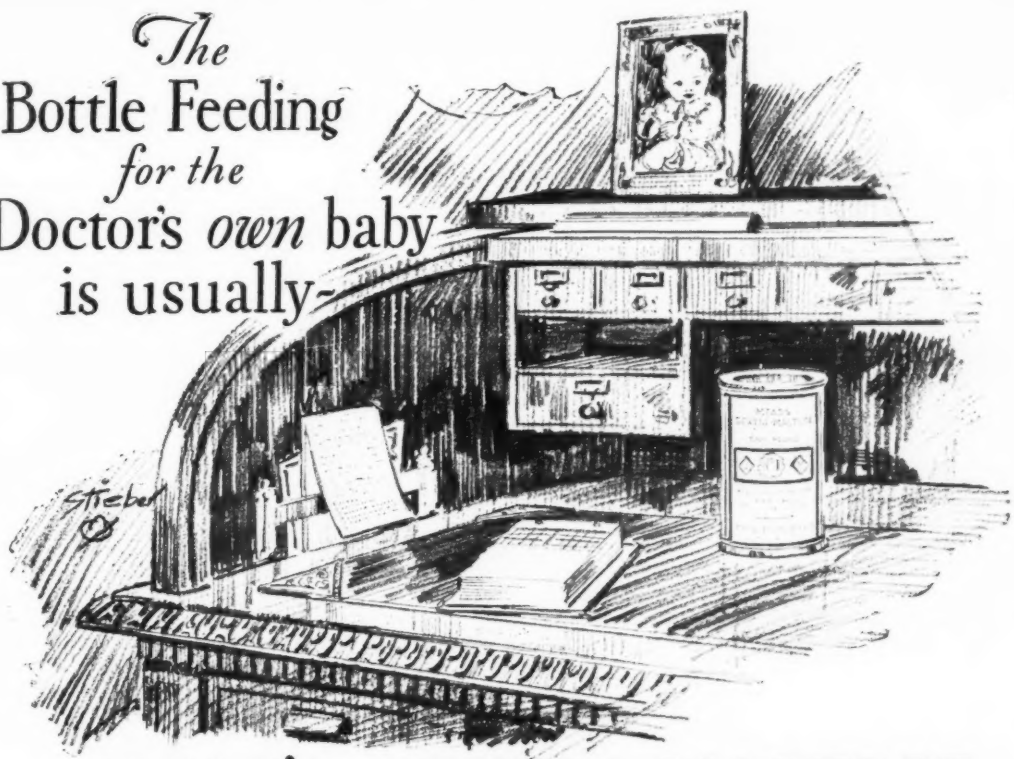

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STATE NEWS ITEMS

The Panama City Hospital, Inc., which operates under the supervision of the Bay County Medical Society, recently elected the following officers: Dr. D. M. Adams, president; Dr. J. M. Nixon, secretary; Miss Dorothy L. Gadwell, treasurer and superintendent. The following constitutes the staff of this hospital: J. M. Nixon, chief of surgery; J. M. Whitfield, X-ray, skin and physiotherapy; W. J. Blackshear, anesthetics; W. J. Lee, diseases of rectum; D. M. Adams, nervous diseases.

* * *

At the December meeting of the Pasco-Hernando-Citrus County Medical Society, the following officers were elected: George A. Dame, Inverness, president; W. S. Hancock, Jr., Brooksville, vice-president; R. D. Sistrunk, Dade City, vice-president; and T. F. Jackson, Dade City, secretary and treasurer. The annual banquet of this Society was held at the Tangerine Hotel, Brooksville, January 12th. There was a good attendance and an excellent program was enjoyed. Among the visiting doctors were Drs. J. C. Chandler, and William Blake of Tampa and Herman Watson of Lakeland.

* * *

Dr. John S. Helms of Tampa was reelected chairman of the Florida Executive Committee for the American College of Surgeons at the conclusion of the two-day session recently held in Tampa.

* * *

At the December meeting of the Marion County Medical Society, the following officers were elected: H. W. Henry, Ocala, president; B. S. Stutts, Dunnellon, vice-president; J. L. Chalker, Ocala, secretary-treasurer. Dr. A. H. Freeman, Ocala, was elected to the censorship committee.

* * *

The annual banquet of the Hillsboro County Medical Society was held January 5th at the Davis Island Country Club. The society was host to the Pasco-Hernando-Citrus County Medical Society.

* * *

The Columbia County Medical Society at its December 19th meeting elected the following officers for 1928: T. H. Bates, president; L. J. Arnold, vice-president; P. C. Farnell, secretary. Dr. L. M. Anderson was elected delegate to the State Association, and Dr. J. H. Dyer, a member of the board of censors. The meeting date was

(*Continued on page 422*)

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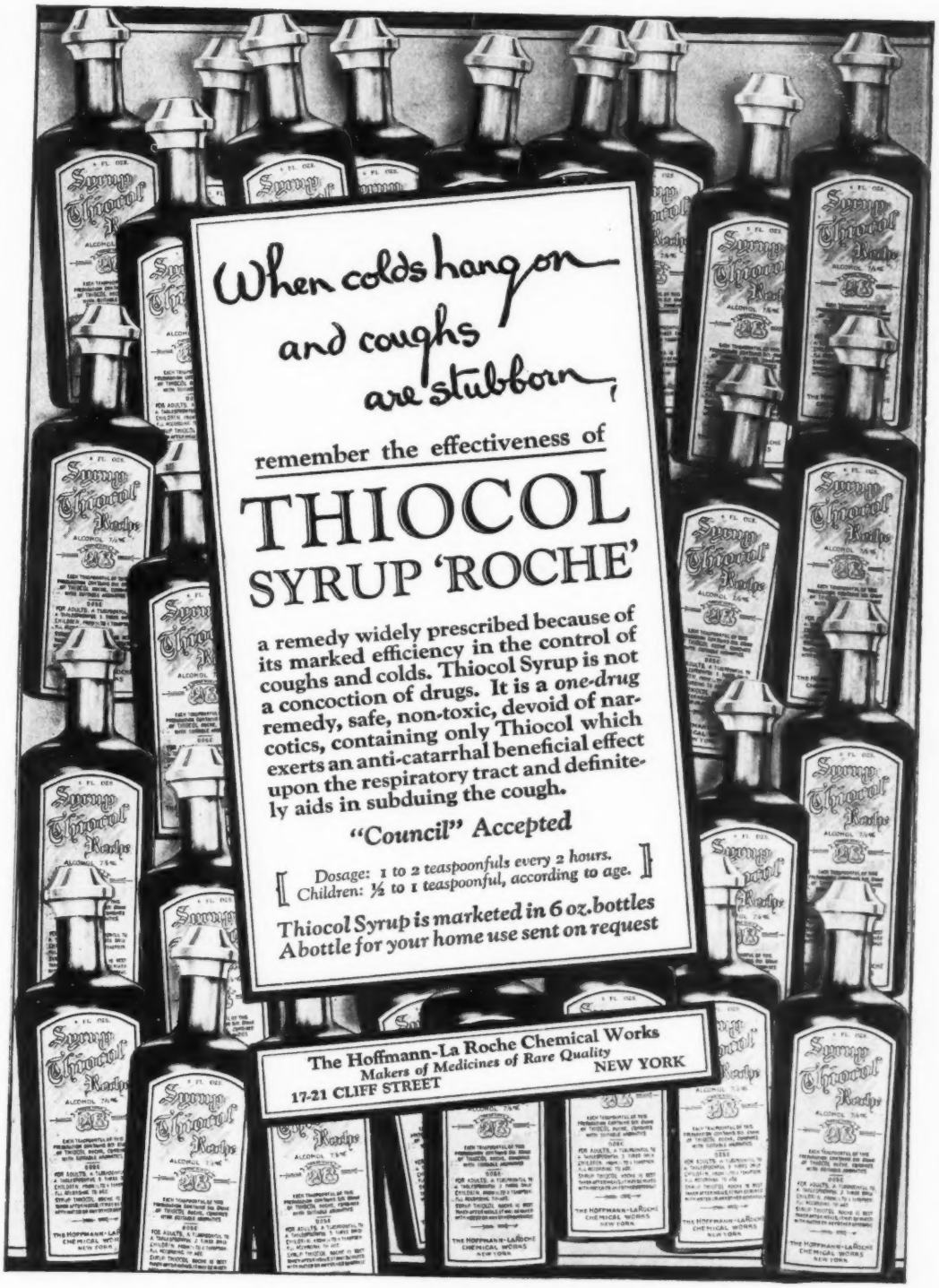
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* * *

The Volusia County Medical Society held its January meeting at the Boathouse Grill in DeLand.

* * *

At the January meeting of the staff of the Munroe Memorial Hospital, Dr. A. H. Freeman was elected Chief of Staff.

* * *

The Jackson County Medical Society met in regular session, January 10th. The following officers were elected for 1928: W. C. Box, Graceville, president; A. A. McKinnon, Marianna, vice-president; C. H. Harrison, Cottondale, secretary-treasurer. Dr. N. A. Baltzell, Marianna, was elected delegate to the State Association.

* * *

Dr. W. S. Nichols of Lake City, former district health officer, is in New Orleans doing special work in the Eye, Ear, Nose and Throat Hospital.

* * *

At the January 20th meeting of the Bay County Medical Society, the following officers were elected: W. J. Lee, Millville, president; D. M. Adams, Panama City, secretary; Drs. W. J. Blackshear, Panama City, W. J. Lee, Millville, and Dr. McGehee of Lynn Haven were elected to the board of censors.

* * *

Dr. Henry Hanson, formerly associated with the State Board of Health, has recently returned to Florida to take his position as field medical officer for the State Board of Health. During Dr. Hanson's absence from the state, he has spent much time abroad and in South America in public health work. His many friends in Florida were delighted to know that he was again making this state his official residence and, furthermore, that he is again identified with the public health work in the state.

* * *

At the recent annual meeting of the Duval County Hospital, the reports showed a very marked increase in the amount of work being done by this hospital. Mr. Richard P. Daniel, chairman of the Duval County Welfare Board, informed the staff that a fifty bed increase in the hospital was contemplated during the year. This will bring the capacity of this hospital to something over two hundred beds.

(*Continued on page 424*)

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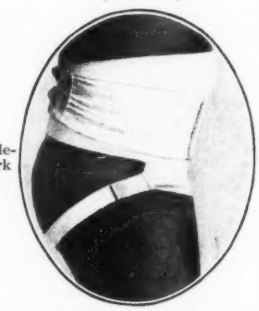

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The Leon-Gadsden-Liberty-Wakulla-Jefferson County Medical Society held its last meeting at the Floridian Hotel, Tallahassee, January 12th at 3 p. m. Dr. H. E. Palmer, president of the Society, presided and the following program was rendered:

"Radium Treatment of Skin and Superficial Cancer," Dr. G. R. Holden, Jacksonville.

"The Nose," Dr. O. G. Kendrick, Taliahassee.

"Toxemias of Pregnancy," Dr. B. M. Rhodes, Tallahassee.

"Some X-ray Diagnoses," Drs. B. A. Wilkinson and F. Clifton Moor.

"The Immunizing of School Children," Dr. A. P. Harrison, Florida State Board of Health.

* * *

The annual sectional meeting of the American College of Surgeons for the states of Florida, Georgia, Alabama, Mississippi and Louisiana was held at the Tampa Bay Hotel in Tampa, January 26th and 27th. Clinics were held at the new Tampa Municipal Hospital each morning. Numerous hospital conferences were held and a municipal health meeting was also held on the evening of the 26th. On the afternoon of the 27th, a scientific program was rendered.

* * *

Dr. R. S. Gill of West Palm Beach and Miss Ruth Bowden of Midland, Georgia, were married at Stuart, Florida, December 12th.

* * *

At the recent annual meeting of the St. Lukes Hospital Association, Jacksonville, Mr. P. L. Gaskins was reelected president of the Association. Mr. Gaskins informed the Association that during the year 1927, there were 43,211 patient days at the hospital. Of this number, 8,406 patient days were for charity and the remaining 34,805 for paid patients. This showed a marked increase in the charity work over the previous years.

* * *

At the January meeting of the Walton-Okaloosa County Medical Society, the following officers were elected: E. P. Webb, Crestview, president; E. D. Thorpe, DeFuniak Springs, vice-president, and A. G. Williams, Lakewood, secretary-treasurer. Drs. G. W. Spires of Darlington and J. J. McGuire of DeFuniak Springs were elected censors.

(*Continued on page 426*)

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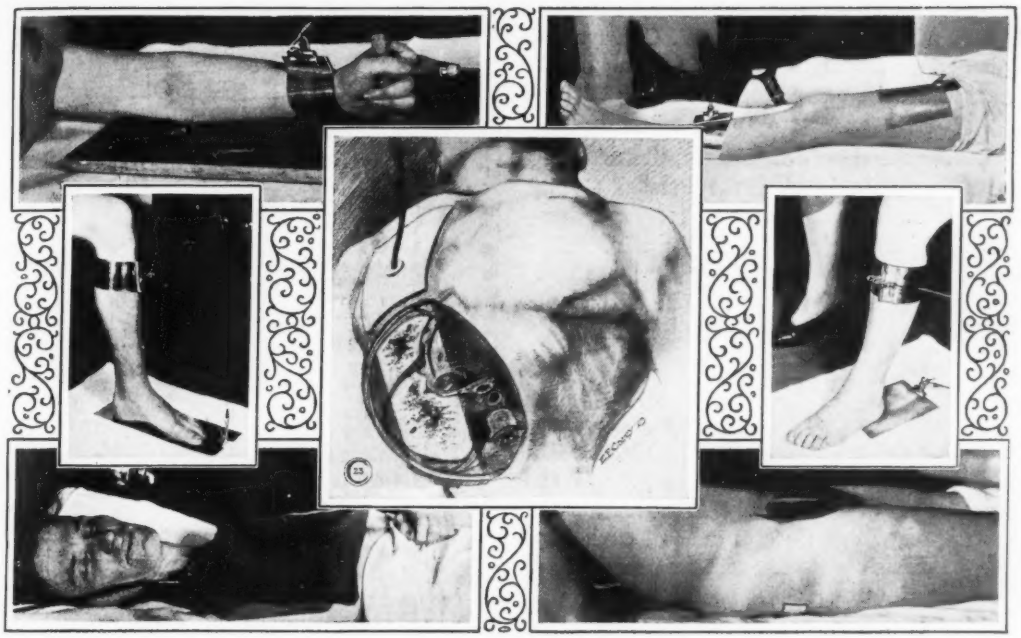

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Drs. M. A. Lischkoff and W. C. Payne of Pensacola attended the Tampa meeting of the American College of Surgeons.

* * *

During the month of January, the following papers were read before the Pinellas County Medical Society:

"Brill's Disease," J. A. Mease, Dunedin.
"Case Report," W. D. Anderson, Largo.
"Glaucoma," M. H. Stuart, St. Petersburg.
"Gallbladder," L. A. Wylie, St. Petersburg.

* * *

Dr. C. T. Nolan of Marietta, Georgia, died while attending the recent meeting of the American College of Surgeons held in Tampa. Dr. Nolan was stricken with angina pectoris and passed away very suddenly. He was an outstanding figure in Georgia medical circles and for the past sixteen years had been secretary of the Board of Medical Examiners.

ADVERTISERS' NOTES.

LISTER'S DISTRIBUTORS.

In this issue appears a two-page colored insert of Lister Bros., Inc., of New York City. For the convenience of readers, a list of their distributors in the field covered by this Journal is herewith given:

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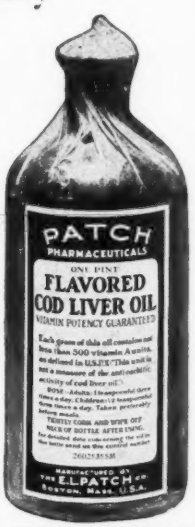

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Bay	D. M. Adams, M.D., Panama City.					71%
Bradford	Seeber King, M.D., Lake Butler.					
Brevard	Geo. W. Wood, M.D., Cocoa.	Varies		Varies		
Broward	Leigh F. Robinson, M.D., Ft. Lauderdale.	2nd Tuesday	8:00 P.M.	Chamber of Commerce	No.	
Columbia.....	P. C. Farnell, M.D., Lake City.	1st Monday.	7:30 P.M.	Chamber of Commerce	No.	100%
Dade	R. M. Harris, M.D., Miami.	1st Friday	8:30 P.M.	Miami City Club	Occasionally.	7%
DeSoto-Hardee-Highlands ...	C. H. Kirkpatrick, M.D., Arcadia.		8:00 P.M.	Varies	No.	70%
Duval	Kenneth A. Morris, M.D., Jacksonville.	1st Tuesday	8:15 P.M.	Arnold-Edwards Auditorium	No.	
Escambia	J. M. Hoffman, M.D., Pensacola.	1st Tuesday	8:00 P.M.	Board of Health Building	No.	
Hamilton	R. A. Barnett, M.D., White Springs.					
Hillsboro	Frank T. Barker, M.D., Tampa.	1st and 3rd Tuesdays	8:00 P.M.	City Hall	No.	
Jackson	C. H. Harrison, M.D., Cottondale.	2nd Tuesday	3:00 P.M.	Marianna	No.	
Lake	W. L. Ashton, M.D., Umatilla.	1st Thursday	12:30 P.M.	Eustis	Yes.	27%
Lee	W. H. Grace, M.D., Ft. Myers.	3rd Friday	7:30 P.M.	Lee Memorial Hospital	No.	
Leon-Gadsden-Liberty-Wakulla-Jefferson.....	F. Clifton Moor, M.D., Tallahassee.	Quarterly	3:00 P.M.	Varies	Yes.	
Madison	Geo. O. Davis, M.D., Madison.					100%
Manatee	J. M. Davis, M.D., Bradenton.	1st and 3rd Tues. Oct. to May; 2nd Tues. May to Oct.	7:00 P.M.	Dixie Grande Hotel	Yes.	
Marion	J. L. Chalker, M.D., Ocala.	3rd Thursday	12:30 P.M.	Harrington Hotel	Yes.	
Monroe	G. R. Plummer, M.D., Key West.	1st Sunday	9:00 P.M.	Varies	Yes.	
Orange	J. R. Chappell, M.D., Orlando.	3rd Wednesday	8:30 P.M.	Varies	No.	
Palm Beach ...	S. W. Fleming, M.D., W. Palm Beach.	2nd Monday	8:00 P.M.	Monterey Hotel	Yes.	23%
Pasco-Hernando-Citrus.....	T. F. Jackson, M.D., Dade City.	2nd Tuesday	8:00 P.M.	Varies	Yes.	40%
Pinellas	O. O. Feaster, M.D., St. Petersburg.	Every other Friday	8:00 P.M.	Fla. Art School	No.	
Polk	Geo. C. Overstreet, M.D., Lakeland.	2nd Wednesday in Feb., Apr., June, Aug., Oct., Dec.	1:00 P.M.	Lakeland	Yes.	
*Putnam	E. W. Warren, M.D., Palatka.					100%
St. Johns	A. C. Walkup, M.D., St. Augustine.	3rd Monday	8:30 P.M.	Varies	Yes.	
St. Lucie-Okeechobee-Indian River-Martin.....	G. C. Hardie, M.D., Ft. Pierce.					10%
Sarasota	F. Metzger, M.D., Sarasota.	2nd Tuesday	8:30 P.M.	Varies	Occasionally.	
Seminole	Chas. Park, M.D., Sanford.					
Sumter	W. E. Mitchell, M.D., Coleman.	2nd Tuesday		Varies	No.	
Suwannee	W. C. White, M.D., Live Oak.					70%
Taylor	R. J. Greene, M.D., Perry.	Last Thursday	12:15 P.M.	Eldorado Cafe	Yes.	
Volusia	R. L. Miller, M.D., Daytona Beach.	2nd Tuesday	7:30 P.M.	Varies	Yes.	33%
Walton-Okaloosa	A. G. Williams, M.D., Lakewood.	3rd Thursday	8:00 P.M.	Varies	Occasionally.	

*Charter pending.

NOTE—(Secretaries: Please submit information to complete the above schedule.)

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